FACAULTY OF PHARMACY NIGER DELTA UNIVERSITY,
WILBERFORE ISLAND, BAYELSA STATE
DEPARTMENT OF CLINICAL SCIENCES AND CLINICAL PRACTICE
PCL 313 CONTINUOUS ASSESSMENT

Disorder 1: **Chronic Obstructive Pulmonary Disease (COPD)**

Definition: Chronic Obstructive Pulmonary Disease (COPD) is a progressive and irreversible lung disease characterized by airflow limitation, making it difficult to breathe. It is a chronic inflammatory condition that affects the airways, leading to a gradual decline in lung function, causing symptoms such as shortness of breath, wheezing, coughing, and chest tightness. The airflow limitation is caused by a combination of airway inflammation, damage to the air sacs, and narrowing of the airways, making it hard for air to flow in and out of the lungs. This progressive disease can severely impact a person's quality of life,making everyday activities challenging, and can also lead to respiratory failure if left untreated or poorly managed.

Etiology/Risk Factors: Long-term exposure to lung irritants like cigarette smoke, air pollution, occupational dusts, and genetic factors (e.g., alpha-1 antitrypsin deficiency).

Types: Emphysema, Chronic Bronchitis, and Refractory (non-reversible) Asthma.

Signs/Symptoms

-Shortness of breath (dyspnea),Wheezing,Coughing (with or without mucus production),Chest tightness,Blue lips or fingers (in severe cases, indicating low oxygen levels),Fatigue,Weight loss,Frequent respiratory infections,Rapid breathing rate,Heart palpitations,Swelling in legs, ankles, and feet (due to fluid buildup),Confusion, memory loss, and difficulty concentrating (in severe cases, due to low oxygen

Pathophysiology:

Chronic Obstructive Pulmonary Disease (COPD) is characterized by a complex interplay of airway inflammation, lung tissue damage, and loss of alveolar surface area, leading to irreversible airflow limitation. The pathophysiological mechanisms involve:

1. Airway Inflammation: Chronic exposure to noxious particles and gases triggers an inflammatory response in the airways, leading to the activation of immune cells, release of inflammatory mediators, and increased mucus production.
3. Lung Tissue Damage: Destruction of lung tissue, particularly in the alveoli, leads to a decrease in the surface area available for gas exchange.
4. Emphysema: Enlargement of airspaces and destruction of alveolar walls result in a loss of lung elasticity and recoil, making it difficult for air to leave the lungs.
5. Chronic Bronchitis: Inflammation and scarring of the airways lead to a decline in lung function.
6. Loss of Alveolar Surface Area: Destruction of alveoli reduces the surface area for gas exchange, making it harder for oxygen to enter the bloodstream and carbon dioxide to be removed.

Diagnosis/ Differential Diagnosis: Spirometry, chest X-ray, CT scan, arterial blood gas analysis, and ruling out other conditions like asthma, heart failure, and pneumonia.

Disorder 2:**Pneomonia**

Definition: Pneumonia is a serious and potentially life-threatening infection that inflames the air sacs (alveoli) in the lungs, making it difficult to breathe and leading to impaired gas

exchange.
This inflammatory condition causes the air sacs to fill with pus, fluid, and cellular debris, reducing the lungs' ability to expand and take in oxygen. Pneumonia can be caused by various microorganisms, including bacteria, viruses, fungi, and parasites, and can affect one or both lungs.
The infection can range from mild to severe, with symptoms worsening over time if left untreated. In severe cases, pneumonia can lead to respiratory failure, acute respiratory distress syndrome (ARDS), and even death.

Etiology/Risk Factors: Bacterial (e.g., Streptococcus pneumoniae), viral (e.g., influenza), or fungal infections, and risk factors like age, compromised immune system, and underlying medical conditions.

Types: Community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP), and ventilator-associated pneumonia (VAP).

Signs/ Symptoms: Cough, fever, chills, shortness of breath, chest pain, fatigue, and confusion (in severe cases).

Pathophysiology: Inflammation and fluid buildup in the alveoli, leading to impaired gas exchange.

Diagnosis/Differential Diagnosis: Chest X-ray, CT scan, blood tests, sputum analysis, and ruling out other conditions like bronchitis, asthma, and pulmonary embolism.

Adapted Cells

Adapted cells are specialized cells that have undergone changes in their structure and function to survive and thrive in a specific environment.
Adapted cells can be found in various tissues and organs, and their adaptations can be driven by factors such as environmental pressures,genetic mutations,epigenetic changes,Cellular interactions
Examples of adapted cells include:
- Immune cells that adapt to recognize and fight pathogens
- Stem cells that differentiate into specialized cell types
- Cells that adapt to high-altitude or high-stress environments
disease mechanisms, and potential therapeutic targets.

Normal cell

A normal cell, also known as a healthy or typical cell, is a functional unit of life that maintains homeostasis and performs its intended biological role. Normal cells are essential for the proper functioning of tissues, organs, and systems, and their integrity is crucial for maintaining overall health and well-being.

Characteristics of a normal cell include:
- Integrity of cell membrane and organelles
- Balanced metabolism and energy production
- Proper DNA replication and cell division
- Effective communication with neighbouring cells
- Ability to adapt to changes in environment
- Maintenance of cellular shape and structure
- Functioning apoptotic mechanisms (programmed cell death)

Cell Injury:

Cell injury occurs when a cell is damaged by a physical, chemical, or biological stressor, disrupting its

normal functioning. This can lead to changes in cellular structure, function, or both. Causes of cell injury:Hypoxia (lack of oxygen),Toxins and poisons,Infections,Physical trauma,Nutritional deficiencies,Metabolic disorders,Cell injury can lead to various diseases and conditions, including cancer,neurodegenerative disorders, and organ failure. Understanding cell injury mechanisms is crucial for developing treatments and therapies to prevent or repair cellular damage.

Cell Death

Cell death, also known as cellular demise or mortality, is the process by which a cell ceases to function and ultimately dies

Cell death can be triggered by factors such as:
DNA damage,Oxidative stress,Infection,Toxins,Nutrient deprivation,Hypoxia (lack of oxygen). It also plays a crucial role in,eliminating damaged or dysfunctional cells,maintaining tissue homeostasis,responding to stress and injury

The Eicosanoid pathway

The Eicosanoid biosynthetic cascade is a intricate biochemical network that plays a pivotal role in modulating the inflammatory response, mediated by the enzymatic conversion of arachidonic acid into a plethora of bioactive lipid mediators, including prostaglandins, thromboxanes, leukotrienes, and lipoxins.

This complex pathway is tightly regulated by a series of enzymes, including phospholipase A2, cyclooxygenase (COX)-1 and COX-2, and 5-lipoxygenase, which orchestrate the production of pro-inflammatory and anti-inflammatory eicosanoids, thereby modulating the immune response and inflammation.

Dysregulation of the Eicosanoid pathway has been implicated in a myriad of inflammatory disorders, including rheumatoid arthritis, asthma, atherosclerosis, and neoplastic diseases. Pharmacological modulation of this pathway, via the administration of nonsteroidal anti-inflammatory drugs (NSAIDs), selective COX-2 inhibitors, and leukotriene receptor antagonists, can effectively mitigate inflammation and alleviate symptoms.

Discuss hypoxia in details as the cause of cell injury

Hypoxia is a state of inadequate oxygen supply to cells and tissues, leading to cellular injury and potentially, cell death. It is a critical cause of cell injury and can occur due to various factors.

Types of Hypoxia:

1. Hypoxic hypoxia (inadequate oxygen supply)
2. Anemic hypoxia (reduced oxygen-carrying capacity)
3. Stagnant hypoxia (impaired blood flow)
4. Cytotoxic hypoxia (impaired oxygen utilization)

Causes of Hypoxia:

1. Respiratory failure
2. Cardiac failure
3. Anemia
4. Carbon monoxide poisoning

5. High altitude
6. Smoke inhalation
7. Shock
8. Tumors

Effects of Hypoxia on Cells:
1. Impaired ATP production
2. Increased lactate production
3. Cellular acidosis
4. Disrupted calcium homeostasis
5. Activation of stress responses (e.g., HIF-1α)
6. DNA damage
7. Inflammation

Cellular Responses to Hypoxia:
1. Adaptation (e.g., increased glucose uptake)
2. Angiogenesis (new blood vessel formation)
3. Apoptosis (programmed cell death)
4. Necrosis (unprogrammed cell death)

Consequences of Prolonged Hypoxia:
1. Organ dysfunction
2. Cell death
3. Tissue damage
4. Increased risk of infections
5. Impaired wound healing

Clinical Examples:
1. Myocardial infarction (heart attack)
2. Stroke
3. Chronic obstructive pulmonary disease (COPD)
4. Cancer
5. Sepsis

Understanding hypoxia's mechanisms and effects is crucial for developing therapeutic strategies to prevent or mitigate cell injury and promote tissue recovery.
Outline the main adaptive respond mechanism which could result when cells are exposed to internal/ external stresses
Cells have evolved various adaptive response mechanisms to cope with internal and external stresses. Here's an outline of the main adaptive response mechanisms:
I. Cellular Stress Response Pathways

1. Heat Shock Response (HSR)
2. Unfolded Protein Response (UPR)
3. DNA Damage Response (DDR)

4. Oxidative Stress Response (OSR)
5. Hypoxia-Inducible Factor (HIF) Pathway

II. Molecular Chaperone Activation
1. Heat Shock Proteins (HSPs)
2. Glucose-Regulated Proteins (GRPs)
3. Calnexin/Calreticulin
III. Cell Cycle Regulation
1. Cell cycle arrest
2. DNA repair
3. Apoptosis (programmed cell death)
IV. Metabolic Adaptations
1. Autophagy
2. Mitophagy (selective mitochondrial degradation)
3. Glycolysis enhancement
4. Glucose metabolism reprogramming
V. Epigenetic Modifications
1. Histone modifications
2. DNA methylation
3. Chromatin remodeling
VI. Signaling Pathway Activation
1. MAPK (Mitogen-Activated Protein Kinase) pathways
2. PI3K/AKT (Phosphatidylinositol 3-Kinase/Protein Kinase B) pathway
3. NF-κB (Nuclear Factor kappa-light-chain-enhancer of activated B cells) pathway

VII. Anti-Apoptotic Mechanisms
1. Bcl-2 family proteins
2. Inhibitor of Apoptosis Proteins (IAPs)
3. FLICE-like inhibitory protein (FLIP)

VIII. Inflammatory Response
1. Cytokine production
2. Chemokine production
3. Immune cell recruitment
When cells are exposed to internal or external stresses, these adaptive response mechanisms help maintain cellular homeostasis, prevent damage, and promote survival.

The various inflammatory mediators and at what point do we apply NSAIDs to aid or support inflammation Inflammatory mediators are chemical signals that promote inflammation. Here's an overview:

Initial Phase (0-4 hours)
1. Histamine (vasodilation, increased permeability)
2. Bradykinin (vasodilation, pain)
3. Serotonin (vasodilation, pain)

4. Prostaglandins (PGs) - PGE2, PGF2α (pain, inflammation)

Acute Phase (4-48 hours)
1. Cytokines:
   - TNF-α (tumor necrosis factor-alpha)
   - IL-1β (interleukin-1 beta)
   - IL-6 (interleukin-6)
2. Chemokines:
   - CXCL8 (interleukin-8)
   - CCL2 (monocyte chemoattractant protein-1)
3. Adhesion molecules:
   - ICAM-1 (intercellular adhesion molecule-1)
   - VCAM-1 (vascular cell adhesion molecule-1)

Chronic Phase (beyond 48 hours)
1. Cytokines:
   - IL-4 (interleukin-4)
   - IL-13 (interleukin-13)
   - TGF-β (transforming growth factor-beta)
2. Growth factors:
   - VEGF (vascular endothelial growth factor)
   - PDGF (platelet-derived growth factor)

NSAIDs Application

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are used to aid or support inflammation during the:
1. Acute phase (4-48 hours): NSAIDs can help reduce inflammation, pain, and fever.
2. Chronic phase (beyond 48 hours): NSAIDs may be used to manage chronic inflammation, but long-term use can have adverse effects.

NSAIDs Mechanism
NSAIDs inhibit:
1. Cyclooxygenase (COX) enzymes:
   - COX-1: involved in physiological processes
   - COX-2: involved in inflammatory responses
2. Prostaglandin synthesis:
   - Reduce PGE2, PGF2α, and TXA2 production

Examples of NSAIDs
1. Ibuprofen (Advil, Motrin)
2. Naproxen (Aleve)
3. Aspirin

4. Celecoxib (Celebrex)
5. Diclofenac (Voltaren)

When to Apply NSAIDs
1. Acute injuries (e.g., sprains, strains)
2. Post-surgical inflammation
3. Chronic inflammatory conditions (e.g., arthritis, tendinitis)
4. Fever reduction

Contraindications and Precautions
1. Bleeding disorders
2. Gastrointestinal issues (ulcers, bleeding)
3. Renal impairment
4. Cardiovascular disease
5. Pregnancy and lactation

It's essential to consult a healthcare professional before using NSAIDs, especially for prolonged periods or in combination with other medications.

Outcomes of inflammation resolutions
3. Type I and Type II inflammation

Here's an overview of the outcomes of inflammation resolution, Type I and Type II inflammation:

Outcomes of Inflammation Resolution
1. Tissue repair and regeneration
2. Restoration of tissue function
3. Elimination of infectious agents or harmful stimuli
4. Return to homeostasis
5. Resolution of clinical symptoms (e.g., pain, swelling, redness)

Type I Inflammation
(Also known as acute or classical inflammation)
Characteristics:
1. Immediate response to infection or injury
2. Neutrophil-dominated response
3. Pro-inflammatory cytokines (TNF-α, IL-1β, IL-6)
4. Activation of complement system
5. Increased vascular permeability

Examples:
1. Bacterial infections (e.g., pneumonia, sepsis)
2. Viral infections (e.g., influenza, HIV)
3. Acute injury (e.g., cuts, burns)
4. Autoimmune diseases (e.g., rheumatoid arthritis)

Type II Inflammation

(Also known as chronic or delayed-type hypersensitivity)
Characteristics:
1. Delayed response to antigen or injury
2. Mononuclear cell-dominated response (macrophages, T cells)
3. Cytokines: IL-4, IL-13, TGF-β
4. Activation of immune cells (T cells, macrophages)
5. Tissue damage and fibrosis

Examples:
1. Chronic infections (e.g., tuberculosis, leprosy)
2. Autoimmune diseases (e.g., multiple sclerosis, lupus)
3. Allergic reactions (e.g., asthma, atopic dermatitis)
4. Cancer

Key differences between Type I and Type II inflammation:
1. Time course: Type I is immediate, while Type II is delayed.
2. Cell types: Type I involves neutrophils, while Type II involves mononuclear cells.
3. Cytokine profiles: Type I is pro-inflammatory, while Type II is more regulatory.
4. Tissue damage: Type I is typically reversible, while Type II can lead to chronic damage.
Understanding these differences is crucial for developing targeted therapeutic strategies.
a. List 3 Factors affecting Respiratory System Disorders b. List the three main types of Respiratory System Disorders
Factors affecting Respiratory System Disorders:

1. Environmental factors (air pollution, smoking)
2. Genetic predisposition
3. Lifestyle factors (obesity, physical inactivity)
Main types of Respiratory System Disorders:
1. Obstructive lung diseases (COPD, asthma)
2. Restrictive lung diseases (pulmonary fibrosis)
3. Infectious diseases (pneumonia, tuberculosis)
Major difference between Asthma and COPD pathophysiology:
Asthma: reversible airway constriction, inflammation, and hyperresponsiveness
COPD: irreversible airway damage, chronic inflammation, and airflow limitation

Section B
2d. Causes of genitourinary disorders:
1. Bacterial infections (UTIs)
2. Kidney stones
3. Hormonal imbalances (e.g., diabetes)

Case Study (Ebiere)
Initial treatment plan:
1. Ascites: Spironolactone (continue), fluid restriction, paracentesis
2. Nausea and vomiting: Antiemetics (e.g., metoclopramide), lactulose dose adjustment

3. Confusion: Address electrolyte imbalance (hyponatremia), lactulose dose adjustment, consider thiamine supplementation

Section C
2a. Compare and contrast negative (-ve) and positive (+ve) feedback mechanisms:
-ve feedback: maintains homeostasis, reverses changes (e.g., insulin regulation)
+ve feedback: amplifies changes, promotes further response (e.g., blood clotting)

2b. Regulation of:
1. Calcium level: Parathyroid hormone (PTH) and vitamin D (-ve feedback)
2. Cortisol level: Hypothalamic-pituitary-adrenal axis (-ve feedback)
3. Lactation: Oxytocin and prolactin (+ve feedback)
Section D
1. General treatment modalities for infections:
2. Antibiotics
3. Antivirals
4. Antifungals
5. Supportive care (hydration, rest)
Section E
1. Host-tumor interrelation and clinical relevance:

Tumor cells interact with host cells, influencing growth, invasion, and metastasis. Understanding this interrelation informs cancer diagnosis, treatment, and prevention strategies.

Section A (continued)
(a) Define heart failure:
Heart failure: inability of the heart to pump blood efficiently, meeting the body's needs.
(b) Clinical features and risk factors:

Clinical features:
1. Shortness of breath
2. Fatigue
3. Swelling (edema)

Risk factors:
1. Hypertension
2. Coronary artery disease
3. Diabetes

(c) Differential diagnosis for heart failure:
1. Pulmonary embolism
2. Chronic obstructive pulmonary disease (COPD)
3. Cardiac tamponade
4. Hypothyroidism

Section B (continued)
(a) Risk factors of Hypertension:
1. Family history
2. Obesity
3. Physical inactivity
4. High sodium intake
5. Age

(b) Morphological types of necrosis:
1. Coagulative necrosis
2. Liquifactive necrosis
3. Caseous necrosis
(c) Effects of Atherosclerotic disease of the coronary artery:

1. Myocardial infarction
2. Heart failure
3. Arrhythmias
4. Sudden cardiac death
Ia. List the cardinal signs of Inflammation
Ib. Briefly discuss ACUTE Inflammation

Write a short note on FIVE of the following clinical cases Emphysema,
Interstitial cystitis (IC), Prostatitis, Kidney stones, and Occupational respiratory disorders
a. What is an infectious disease and state the difference between infectious diseases and non-infectious diseases

b. List the agents that cause infectious discases and list two diseases they cause
a. Describe the endocrine system and state five major functions of the endocrine system
b. List seven organs/glands of the endocrine system and state one function each they perform

A 70-year-old male presents with acute fightness of the chest, fatigue, lightheadedness, anxiety, and chest pain.
State and define the diagnosis.
What is the leading cause of this condition?
b. Give the Diagnosis and define
c. Give a detailed account of the leading cause of coronary artery disease.

Define Cancer
B. Briefly describe the systematic effects of cancer
A. Differentiate between gastric ulcer disease and duodenal ulcer disease

B. What are the basic preventive measures against ulcer disease?

ANSWERS:
Here are the detailed answers:

1a. Cardinal signs of Inflammation:
1. Redness (rubor)
2. Swelling (tumor)
3. Heat (calor)
4. Pain (dolor)
5. Loss of function (functio laesa)

1b. Acute Inflammation:
Acute inflammation is a short-term, immediate response to tissue damage or infection, characterized by increased blood flow, vasodilation, and recruitment of white blood cells.

Clinical Cases:
1. Emphysema: Chronic obstructive pulmonary disease (COPD) characterized by air sac damage and shortness of breath.
2. Interstitial cystitis (IC): Bladder inflammation causing pelvic pain, urinary frequency, and urgency.
3. Prostatitis: Inflammation of the prostate gland, causing pelvic pain, urinary symptoms, and painful ejaculation.
4. Kidney stones: Mineral deposits causing urinary obstruction, severe pain, and nausea.
5. Occupational respiratory disorders: Lung diseases caused by workplace exposures (e.g., asbestos, silica).
2a. Infectious disease:
An infectious disease is a condition caused by pathogens (bacteria, viruses, fungi, or parasites) that enter and multiply within the body.
Difference: Infectious diseases are caused by pathogens, whereas non-infectious diseases are caused by genetic, environmental, or lifestyle factors.

2b. Agents causing infectious diseases:
1. Bacteria (e.g., Tuberculosis, Pneumonia)
2. Viruses (e.g., Influenza, HIV)
3. Fungi (e.g., Candidiasis, Aspergillosis)
4. Parasites (e.g., Malaria, Toxoplasmosis)

3a. Endocrine system:
The endocrine system is a network of glands producing hormones regulating various bodily functions.

Five major functions:

1. Growth and development
2. Metabolism
3. Electrolyte balance
4. Reproduction
5. Stress response

3b. Organs/glands of the endocrine system:
1. Pituitary gland (regulates hormone production)
2. Thyroid gland (regulates metabolism)
3. Adrenal glands (regulate stress response)
4. Pancreas (regulates blood sugar)
5. Ovaries (regulate female reproductive hormones)
6. Testes (regulate male reproductive hormones)
7. Hypothalamus (regulates body temperature)

Case Study:
Diagnosis: Acute Coronary Syndrome (ACS)
Leading cause: Coronary artery disease (atherosclerosis)

1. Definition of Cancer:
Cancer is a group of diseases characterized by uncontrolled cell growth, invasion, and metastasis.
Systematic effects of cancer:
1. Weight loss
2. Fatigue
3. Pain
4. Infections
5. Organ dysfunction

5a. Differentiate between gastric ulcer disease and duodenal ulcer disease:
Gastric ulcers:
- Located in the stomach
- Associated with Helicobacter pylori infection
- More common in older adults

Duodenal ulcers:
- Located in the duodenum (first part of small intestine)
- Associated with Helicobacter pylori infection and acid secretion
- More common in younger adults
5b. Preventive measures against ulcer disease:
1. Helicobacter pylori eradication
2. Acid-reducing medications
3. Avoid NSAIDs and aspirin
4. Maintain healthy diet and lifestyle

5. Manage stress
Briefly discuss paraneoplastic syndrome in cancer. Give examples
Briefly describe and give 2 examples each
(1) Autoimmune discase
Immunodeficiency disease
Write short note on
Sinusitis
Lung cancer
Emphysema
Urinary tract infections
Ganglionic, Shaft
Interstitial cystitis
Prostatitis
a. Highlight the morphologic types of Necrosis.
State the purpose of inflammation.
6 a. What are the various changes that can occur in a cell?
b. Highlight the mechanism of cell injury.

Outline the progressive stages of alcoholic liver disease/ What are the Manifestations of cirrhosis and liver failure?
Here are the detailed answers:
Paraneoplastic Syndrome
Paraneoplastic syndromes are rare disorders caused by cancer, but not directly related to tumor growth or metastasis. Examples:
1. Lambert-Eaton syndrome (muscle weakness, associated with small-cell lung cancer)
2. Hypercalcemia (elevated calcium levels, associated with various cancers)
3. Thrombophlebitis migrans (blood clots, associated with pancreatic cancer)
4. Cushing's syndrome (hormonal imbalance, associated with lung, thyroid, or adrenal cancers)

Autoimmune Diseases
Autoimmune diseases occur when the immune system attacks healthy tissues.
Examples:
1. Rheumatoid arthritis (joint inflammation)
2. Systemic lupus erythematosus (multi-organ inflammation)

Immunodeficiency Diseases
Immunodeficiency diseases occur when the immune system is impaired.

Examples:
1. HIV/AIDS (human immunodeficiency virus/acquired immunodeficiency syndrome)
2. Severe combined immunodeficiency (SCID)

Short Notes

1. Sinusitis: Inflammation of sinuses, often caused by infection.
2. Lung cancer: Uncontrolled cell growth in lung tissue.
3. Emphysema: Chronic obstructive pulmonary disease (COPD) characterized by air sac damage.
4. Urinary tract infections (UTIs): Bacterial infections in the urinary system.
5. Ganglionic, Shaft: Not a medical term.
6. Interstitial cystitis: Bladder inflammation causing pelvic pain and urinary symptoms.
7. Prostatitis: Inflammation of the prostate gland.

Morphologic Types of Necrosis
1. Coagulative necrosis
2. Liquifactive necrosis
3. Caseous necrosis
4. Fat necrosis
5. Fibrinoid necrosis

Purpose of Inflammation
Inflammation aims to:
1. Eliminate harmful stimuli (pathogens, toxins)
2. Repair damaged tissue
3. Restore tissue function

Cell Changes
Cells can undergo:
1. Adaptation (reversible changes)
2. Injury (reversible or irreversible)
3. Death (apoptosis or necrosis)
4. Transformation (cancerous changes)
5. Proliferation (increased cell growth)

Mechanism of Cell Injury
Cell injury occurs due to:
1. Hypoxia (oxygen deficiency)
2. Toxins (chemical or biological)
3. Infection
4. Physical damage (trauma, radiation)
5. Metabolic disturbances

Alcoholic Liver Disease
Progressive stages:
1. Fatty liver
2. Alcoholic hepatitis
3. Fibrosis

4. Cirrhosis

Manifestations of cirrhosis and liver failure:
1. Jaundice
2. Ascites
3. Hepatic encephalopathy
4. Bleeding disorders
5. Liver cancer

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