Learning Objectives

• Outline the structure of the immune system.
• Describe the antigen-antibody response.
• Distinguish between natural and acquired immunity.

Learning Objectives

• Differentiate between a normal immune response and an allergic reaction.
• Distinguish between the four types of hypersensitivity reaction.
• Describe signs and symptoms and management of local allergic reactions based on an understanding of the pathophysiology associated with this condition.

Learning Objectives

• Identify allergens associated with anaphylaxis.
• Describe the pathophysiology, signs and symptoms, and management of non-systemic allergic reaction.
• Describe the pathophysiology, signs and symptoms, and management of anaphylaxis.
Immune System Overview

- Complex network of cells, tissues, organs
  - Work together to protect body against “attacks” by foreign substances
  - Most are pathogenic microbes or pathogens
- Examples
  - Bacteria
  - Parasites
  - Fungi
  - Viruses that can cause infection

Immune System Overview

- Primary role
  - Prevent these foreign substances from entering body
  - If fails, launch attack so foreign bodies are found and destroyed
- Organs of immune system include spleen, tonsils, adenoids, lymph nodes, thymus
  - Organs are positioned throughout body
  - Important outposts for lymphocytes—“key players” in immune system
Lymphocyte

- Fundamental cellular unit of immune system
- 25 percent of circulating white blood cells are lymphocytes
- Divided into two major classes
  - B lymphocytes (B cells)
  - T lymphocytes (T cells)
  - Roles are different and complimentary

Lymphocyte

- B cells produce antibodies
  - Antibodies are proteins, not cells
  - "Magic bullets" that seek out specific invaders (antigens)
  - Antigens have marker molecules that identify them as foreign
  - When found, antibodies trigger process that destroys them
  - Antibodies in blood and lymph make up humoral immunity
    - Humoral immunity responds to antigens, such as bacteria and foreign tissue

Lymphocyte

- T cells
  - Respond only to specific organisms
  - Three varieties of T cells
    - Killer T cells (TK cells): attack invading organism with chemicals
    - Helper T cells (TH cells): encourage B cells to produce antibodies
    - Suppressor T cells (TS cells): help regulate immune response to protect body from its own defense
Lymphocyte

- T cells
  - Work of T cells is called cell-mediated immunity
    - Does not involve antibodies
    - Activates lymphocytes that attack and destroy foreign material

Natural and Acquired Immunity

- Once B cells and T cells have been activated by antigen, some become memory cells
  - Many memory cells take up permanent residence in lymph nodes, GI tract, and spleen
  - Others travel through lymphatic system and bloodstream
  - There they join with other lymphocytes and remain on guard for their chosen antigen
Natural and Acquired Immunity

• Memory cells
  – Ensure that next time body is exposed to same antigen that produced memory cell, immune system will be set into motion to destroy it
    • Known as immunologic memory
    • Process by which this occurs is known as immunity
  – Immunity can be either natural or acquired

Based on the list of allergens in Box 27-2, what are some likely dispatch locations where you might be expected to care for a patient who is experiencing an allergic reaction?

Natural Immunity

• Also known as innate immunity or nonspecific immunity
  – “Naturally” exists
  – Not antigen specific
    • Does not require previous exposure to antigen
  – Present at birth by way of antibodies newborn receives from mother
    • Antibody IgG travels across placenta and makes newborn immune to same microbes to which mother is immune
    • Children who are nursed also receive IgA from breast milk to protect their stomach
Natural Immunity

• Also known as innate immunity or nonspecific immunity
  – May have heritable component
  – Quick to respond
  – Body's first line of defense against invading organisms

Natural Immunity

• Can be passive or active
• Passive immunity
  – Natural immunity present at birth
  – Can be conveyed through serum obtained from person who is immune to specific infectious agent (artificially acquired passive immunity)
  – Usually provides protection for only a few weeks
  – Carries risk of causing sensitivity reactions

Natural Immunity

• Active immunity
  – Can be triggered by both infection and vaccination
  – Vaccinations given with inactive (noninfectious) pathogens also provide active immunity
  – Usually is short-lived
Acquired Immunity

• Also known as adaptive immunity
  – Develops after exposure to specific antigens
  – Is “acquired” following B cell and T cell activation, which creates immunologic memory
  – May occur through immunization with vaccine that contains weak form of specific antigen
  – Generally long-term, often considered permanent

Acquired Immunity

• Slower to respond than natural immunity
  – Immunologic memory significantly improves rate of response to subsequent exposure to same antigen
• Acts as body’s second line of defense against invading organisms
• Can be passive (acquired by transfer of antibodies) or active (through immunization)
Immune Response

• First two lines of defense against infection use same mechanism to respond to all pathogens
  — Immune response is specific to individual pathogens

Immune Response

• Immune system that makes up immune response has four unique characteristics
  — Has “self-nonself” recognition, usually responds only to foreign antigens
  — Produces antibodies that are antigen specific
    • New antibodies can be produced in response to new antigens
  — Memory cells produced by some of antibody-producing lymphocytes allow for more rapid response to repeat invasions by same antigen

Immune Response

• Immune system that makes up immune response has four unique characteristics
  — Immune system is self-regulated
    • Activates only when pathogen invades
    • Prevents healthy tissues from being destroyed
    • When function goes awry, allergic reactions and autoimmune disease can occur
    • Immune system also may require extrinsic regulation with drugs in patients with transplanted organs or severe autoimmune diseases
What major immune disorder causes life-threatening airway, breathing, and circulatory problems?

Allergic Reactions

- Antigens can enter the body exogenously (from outside) by injection, ingestion, inhalation, or absorption
  - Stimulate immune system to produce antibodies
  - Aid in neutralizing antigens and removing them from body
  - This normal antigen-antibody reaction protects body from disease by activating immune response

Allergic Reactions

- Immune responses normally are protective
  - Can become oversensitive
  - Can become directed toward harmless antigens to which people often are exposed (e.g., ragweed and pollen)
  - When occurs, response is termed allergic
  - Antigen or substance causing allergic response is called allergen
Allergic Reactions

- Common allergens
  - Drugs
  - Insects
  - Foods
  - Latex
  - Animals
  - Pollens and molds

Allergic Reactions

- Also known as hypersensitivity reaction
- Marked by increased physiological response to antigen after previous exposure to same antigen
  - Known as sensitization
  - Starts when circulating antibody combines with specific foreign antigen
    - Results in hypersensitivity reactions
    - Antibodies bound to mast cells or basophils

Allergic Reactions

- Mast cells
  - Cells found in several types of tissues
  - Contain granules that are rich in histamine and heparin
  - Similar to basophils, class of white blood cell that promotes inflammation through release of chemical mediators
- Mast cells and basophils and release of these chemicals play important role in allergic reactions
Allergic Reactions

- Allergic reactions can be mild, moderate, or severe (including anaphylaxis)
  - Mild and moderate reactions, usually are localized affecting skin, upper and lower airways, GI tract
    • Not life threatening
  - Severe allergic reactions affect same body systems
    • May be life threatening

Hypersensitivity Reaction Types

- Type I
  - Anaphylactic or IgE-mediated reactions
  - Anaphylactic reaction is most dramatic
  - May be life-threatening
- Type II
  - Cytotoxic or tissue-specific reactions
- Type III
  - Immune-complex mediated reactions
- Type IV
  - Delayed or cell-mediated reactions
### Localized Allergic Reaction

- Contact with an allergen bridges adjacent antibodies
  - Each antibody binds with invading organism at different site
    - Changes alignment of antibodies on surface of mast cell
    - Causes mast cell to burst and to release active chemical mediators into surrounding fluid
- Localized allergic reactions do not involve entire body
  - Sites of mast cell and basophil mediator release are limited

### Localized Allergic Reaction

- Signs and symptoms of localized allergic reactions
  - Conjunctivitis (inflammation of conjunctiva of eyes)
  - Rhinitis (runny nose)
  - Angioedema (swelling)
  - Urticaria (hives)
  - Pruritus (itching)

### Localized Allergic Reaction

- Best managed with drugs that compete for receptor sites with histamine
  - Prevents histamine from performing its physiological actions
- Common antihistamines
  - Over-the-counter oral and nasal decongestants
  - Prescription and nonprescription diphenhydramine
- Other medications
  - Steroids
  - Topical creams
Anaphylaxis

- Immediate, systemic, life-threatening allergic reaction
  - Associated with major changes in cardiovascular, respiratory, and cutaneous systems
  - Prompt recognition and appropriate drug therapy in prehospital phase are vital to patient survival
  - The term anaphylaxis comes from Greek
    • Means "against or opposite of protection"

Anaphylaxis

- Most extreme form of allergic reaction
  - Accounts for 400 to 800 deaths/year
  - Mortality rate of 3 percent
  - Rapid recognition and aggressive therapy are essential

Which of these effects has the potential to cause death first?
Causative Agents

- Almost any substance can cause anaphylaxis
  - Antigenic agents most frequently associated with anaphylaxis
    - Penicillin (by ingestion or injection)
    - Envenomation by stinging insects
    - Food (especially nuts and shellfish)
  - Regardless of offending antigen, risk of anaphylaxis in sensitive persons increases with each exposure
    - To lesser extent, risk increases with length of exposure or site of inoculation

Anaphylaxis Pathophysiology

- Person first must be exposed to specific antigen to develop hypersensitivity
  - In first exposure, antigen enters body by injection, ingestion, inhalation, or absorption
  - Then activates immune system
  - In susceptible persons, large amounts of IgE antibody are produced
  - IgE antibodies leave lymphatic system and bind to IgE-specific Fc receptors on cell membranes of basophils circulating in blood and to mast cells in tissues surrounding blood vessels

Anaphylaxis Pathophysiology

- Antibodies remain there, are inactive until same antigen is introduced into body a second time
  - With next exposure to specific antigen, allergen cross-links at least two cell-bound IgE molecules
    - Results in degranulation (release of internal substances) of mast cells and basophils and onset of an anaphylaxis
Anaphylaxis Pathophysiology

- Degranulation of target cell is associated with release of pharmacologically active chemical mediators from inside affected basophils and mast cells
  - Chemicals include
    - Histamine
    - Leukotrienes
    - Eosinophil chemotactic factor of anaphylaxis
    - Neutrophils
    - Heparin
    - Kinins
    - Prostaglandins
    - Thromboxanes

- Histamine
  - Protein released by mast cells and basophils
  - Promotes vascular permeability
  - Causes dilation of capillaries and venules and contraction of smooth muscle in GI tract and bronchial tree
Anaphylaxis Pathophysiology

- Histamine
  - Associated increase in gastric, nasal, and lacrimal secretions
    - Results in tearing and rhinorrhea
  - Increased capillary permeability allows plasma to leak into interstitial space
    - Decreases amount of intravascular volume available for heart to pump

- Profound body-wide vasodilation further decreases cardiac preload
  - Decreases stroke volume and cardiac output
    - Leads to flushing, urticaria, angioedema, hypotension
  - Rapid onset of action of histamine
    - Effects are short lived, are quickly broken down by plasma enzymes
Anaphylaxis Pathophysiology

- Leukotrienes
  - Most potent bronchoconstrictors, which cause wheezing
  - Chemical mediators also cause coronary vasoconstriction and increased vascular permeability
  - Formerly were known as slow-reacting substances of anaphylaxis because their effects were delayed relative to histamine
  - Duration of action of these chemicals is much longer than histamine

- Eosinophil chemotactic factor of anaphylaxis
  - Group of active substances released during an anaphylactic reaction
  - Histamine
  - Leukotrienes
- Process of anaphylaxis attracts eosinophils to site of allergic inflammation
  - Eosinophils contain enzyme that can deactivate leukotrienes
Anaphylaxis Pathophysiology

- Remaining chemical mediators (heparin, neutrophils, and kinins) exert varying effects
  - Fever
  - Chills
  - Bronchospasm
  - Pulmonary vasoconstriction

Anaphylaxis Pathophysiology

- Chemical processes can lead rapidly to
  - Upper airway obstruction and bronchospasm
  - Dysrhythmias and cardiac ischemia
  - Circulatory collapse and shock

Assessment Findings

- Differentiate between severe allergic reactions and mimicked anaphylaxis
  - Accurate history and physical assessment necessary
  - Flawed prehospital assessment can have life-threatening consequences
  - Disease entities that may present similar as anaphylaxis
    - Severe asthma with respiratory failure
    - Upper airway obstruction
    - Toxic or septic shock
    - Pulmonary edema (with or without MI)
Assessment Findings

• Signs and symptoms
  – Drug overdose
    • Dystonic reaction to antipsychotics
    • Scombroid poisoning
    • ACE inhibitor angioedema
    • Hypovolemic shock

Assessment Findings

• Initial signs may vary
  – Signs may range from sneezing and coughing to complete airway obstruction (caused by laryngeal and epiglottic edema)
  – Throat tightness and dyspnea
  – Stridor or voice changes
  – Lower airway bronchospasm
  – Associated hypersecretion of mucus caused by actions of histamine, leukotrienes, and prostaglandins may produce wheezing
  – Significant respiratory distress

Cardiovascular Effects

• Cardiovascular manifestations of allergic reactions range from mild hypotension to vascular collapse and profound shock
  – Dysrhythmias (including severe bradycardia)
    • May be related to severe hypoxia and loss of circulating fluid volume
    • Patient may complain of chest pain if myocardial ischemia is present
**GI Effects**

- Anaphylactic reaction may cause
  - Nausea
  - Vomiting
  - Diarrhea
  - Severe abdominal cramping

**GI Effects**

- Increased GI activity is related to
  - Smooth muscle contraction
  - Increased mucous production
  - Outpouring of fluid from gut wall into intestinal lumen initiated by chemical mediators

**Nervous System Effects**

- Responses caused by impaired gas exchange and shock associated with anaphylaxis
  - At first patient may be agitated and speak of sense of impending doom
  - As hypoxia and shock worsen, brain functions deteriorate
    - May result in confusion, weakness, headache, syncope, seizures, and coma
Cutaneous Effects

• Most visible signs that distinguish anaphylaxis from other medical conditions relate to skin
  – Caused by vasodilation induced by histamine release from mast cells
  – Initially patient may complain of warmth and pruritus (itching)
  – Physical examination often reveals diffuse erythema (redness) and urticaria (hives)

Cutaneous Effects

• Hives
  – Well-circumscribed wheals of 1 to 6 cm
  – Can be more reddened or pallid than surrounding skin
  – Often accompanied by severe pruritus

Cutaneous Effects

• Other visible signs
  – Significant swelling of face and tongue and deep tissues (angioedema)
    • Reflects involvement of deeper capillaries of skin and mucous membranes
  – As hypoxia and shock continue, cyanosis may be evident
Primary Survey

• Initial patient care measures directed at providing adequate airway, ventilatory, and circulatory support
• Drug therapy often is definitive treatment initiated as quickly as possible

Primary Survey

• Airway assessment critical
  – Most deaths are related directly to upper airway obstruction
• Conscious patient should be evaluated for:
  – Voice changes
  – Stridor
  – Barking cough
Primary Survey

• Complaints of tightness in neck and dyspnea should alert paramedic of impending airway obstruction
• While drug therapy is underway, airway of unconscious patient should be assessed and secured
  – If air movement is blocked, advance airway should be placed

Primary Survey

• If laryngeal and epiglottic edema are severe, surgical or needle cricothyrotomy
  – Early, elective intubation is indicated for patients with
    • Hoarseness
    • Lingual edema
    • Posterior or oropharyngeal swelling
• If respiratory function deteriorates, medical direction may recommend tracheal intubation (with sedation) without paralytic agents

Primary Survey

• Monitor patient closely for signs of respiratory distress as indicated by
  – Pulse oximetry
  – Capnography
  – Skin color
  – Accessory muscle use
  – Wheezing
  – Diminished breath sounds
  – Abnormal respiratory rates
Primary Survey

• Circulatory status also may deteriorate quickly
  – Pulse quality, rate, and location should be frequently assessed

History

• May be difficult to obtain
  – Critical to rule out other medical emergencies that may mimic anaphylaxis
• Question patient about chief complaint and rapidity of onset of symptoms
• Signs and symptoms usually appear within 1 to 30 minutes of introduction of antigen
  – Onset of reaction can be delayed if exposure is by oral route

History

• Important medical history
  – Previous exposure and response to suspected antigen
  – Identify method of exposure
    • Injection of an antigen often produces most rapid and severe response
  – Chronic or concurrent illness and medication use
  – Preexisting cardiac disease or bronchial asthma
    • Anticipate severe complications as result of allergic reaction
History

• Use of certain drugs may diminish patient’s response to epinephrine and may necessitate administration of other medications
  – Beta-blocking agents
• Determine whether patient has an emergency epinephrine drug kit (e.g., EpiPen), whether medication was administered before EMS arrival

History

• Determine whether patient has taken oral antihistamine or used aerosolized epinephrine
  – Appropriate intervention should not be delayed

How does epinephrine reverse the signs and symptoms of anaphylaxis?
Physical Examination

- Assess vital signs often
  - In severe reactions, most patients initially are
    - Tachycardic
    - Tachypneic
    - Hypotensive
  - Inspect patient’s face and neck for
    - Angioedema
    - Urticaria
    - Tearing
    - Rhinorrhea
  - Note presence of erythema or urticaria on other body regions

Physical Examination

- Assess airway and lung sounds often to evaluate clinical progress of patient
  - Will help paramedic to monitor effectiveness of interventions
  - Cardiac monitoring should be instituted as soon as possible to aid in patient evaluation

Interventions to Prevent Arrest

- Interventions
  - Place patient in position of comfort
    - Elevate legs until replacement fluids improve BP
  - Administer high-concentration O₂
  - Give epinephrine to all patients with clinical signs of shock, airway swelling, or difficulty breathing
    - May be given by IM route
    - IV epinephrine should be considered immediately if life-threatening signs and symptoms present
Interventions to Prevent Arrest

• Interventions
  — Initiate IV therapy with normal saline solution if hypotension is present and does not respond rapidly to epinephrine
    • Rapid infusion of 1 to 2 L (up to 4 L) may be needed initially
  — Position patient recumbent and elevate legs unless precluded by vomiting or shortness of breath
    • Transport
      • Most patients will be observed carefully in hospital for up to 24 hours
      • Many patients do not respond promptly to therapy, symptoms may recur in some patients (biphasic reaction)

Other Drug Therapy

• Additional drug therapy may be helpful
  — Epinephrine is only drug that can reverse life-threatening complications of anaphylaxis immediately
  — Drug of choice
    • Its beta stimulation produces bronchodilation
    • Its alpha stimulation produces vasoconstriction
    • It reduces release of chemical mediators from mast cells

Other Drug Therapy

• Pharmacological agents that may be used with epinephrine
  — Antihistamine to antagonize effects of histamine
  — Beta-agonists to improve alveolar ventilation
  — Corticosteroids to prevent delayed reaction
  — Glucagon (for patients unresponsive to epinephrine, especially those taking antidysrhythmics [beta-blockers])
Other Drug Therapy

• Pharmacological agents that may be used with epinephrine
  – Vasopressors to manage protracted hypotension
  – Albuterol for bronchodilation
  – Diphenhydramine to counter effects of histamine
  – Methylprednisolone to reduce inflammation

Interventions During Arrest

• Cardiac arrest from anaphylaxis may be associated with
  – Profound vasodilation
  – Intravascular collapse
  – Tissue hypoxia
  – Asystole

Interventions During Arrest

• Airway, oxygenation, ventilation
  – Swelling of airway can make bag-mask ventilation and endotracheal intubation difficult or ineffective in patients with anaphylaxis
  – Landmarks for needle cricothyrotomy may not be visible because of severe swelling in soft tissues of the neck
  – Fiberoptic intubation and digital intubation are alternative methods to consider in these situations
  – Smaller than normal endotracheal tubes may be needed because of associated edema
Interventions During Arrest

• Support of circulation
  – Cardiac arrest from anaphylaxis requires
    • Rapid and aggressive volume replacement (2 to 4 L) to support circulation
    • Use of vasopressor drugs to support BP
  – Epinephrine is drug of choice for treatment of vasodilation and hypotension in cardiac arrest
    • Alternative vasoactive drugs may be considered in cardiac arrest secondary to anaphylaxis that does not respond to epinephrine

• Support of circulation
  – May need to administer epinephrine in high doses
  – With asystole or pulseless electrical activity with a heart rate less than 60 beats/minute (most common arrest rhythms in anaphylaxis), administration of atropine should be included
  – Cardiac arrest from anaphylaxis may respond to prolonged periods of CPR
    • Especially when patient is young and has healthy heart and cardiovascular system

Lesson 27.2
Autoimmune Disease, Collagen Vascular Disease, and Organ Transplants
Learning Objectives

- Define autoimmune disease
- Describe the pathophysiology, signs and symptoms, and prehospital considerations for patients who have collagen vascular diseases such as systemic lupus erythematos and scleroderma.
- Identify major complications associated with organ transplant.

Learning Objectives

- List infections associated with organ transplant.
- Outline characteristics of organ rejection.
- Recognize side affects associated with anti-rejection medications.

Collagen Vascular Disease

- Also known as connective tissue disease
  - Collagen is principal structural protein of most body tissues
  - Many connective tissue diseases feature abnormal immune system activity with inflammation in tissues
    - Result of immune system that is directed against one's own body tissues (autoimmune disease)
  - May have both genetic and environmental causes
Collagen Vascular Disease

- Results in accumulation of extra antibodies in circulation
- Diseases
  - Systemic lupus
  - Scleroderma
- In some cases, will be combination of these diseases in same patient
  - Mixed connective tissue disease

Systemic Lupus

- Systemic lupus erythematosus (SLE)
  - Form of disease that most people refer to as “lupus”
  - Usually first affects people between ages of 15 and 45 years
  - Can also occur in childhood or later in life as well

Systemic Lupus

- Immune system produces antibodies against body’s healthy cells and tissues
- With lupus, these antibodies, called autoantibodies, contribute to inflammation of various parts of body and can cause damage to organs and tissues
- Most common type of autoantibody in lupus
  - Antinuclear antibody (ANA)
  - Reacts with parts of cell’s nucleus
Systemic Lupus

- Can affect many parts of the body
  - Joints
  - Skin
  - Kidneys
  - Heart
  - Lungs
  - Blood vessels
  - Brain

Systemic Lupus

- Frequently develop inflammatory heart disease (pericarditis)
- May experience early-onset artherosclerosis as complication of disease

Systemic Lupus

- Most common symptoms
  - Extreme fatigue
  - Painful or swollen joints (arthritis)
  - Unexplained fever
  - Skin rashes
  - Kidney problems
  - Reddish skin rash, called butterfly or malar rash
    - May appear across nose and cheeks
Systemic Lupus

• Systemic effects
  – GI ulceration or hemorrhage, abdominal pain, pancreatitis, cholecystitis, bowel infarction
  – Renal failure
  – Anemia and clotting abnormalities
  – Pericarditis
  – Pleurisy or pleural effusions
  – Skin rash
  – Behavioral changes, seizures, headaches, stroke

Lupus Management

• Symptoms may be mild or serious
• Goal of treatment
  – Prevent flare ups
  – Prevent organ damage and complications
Lupus Management

- Physician care
  - Nonsteroidal anti-inflammatory drugs and corticosteroids to decrease inflammation
  - Antimalarials to treat fatigue, joint pain, skin rashes, and lung inflammation
  - Hormones
  - Diet modification
  - Nutritional supplements
- No cure

Scleroderma

- Derived from Greek words “sklerosis,” meaning hardness, and “derma,” meaning skin
  - Literally means hard skin
- Results from immune system stimulating certain cells (fibroblasts) that cause increased production of collagen
  - Excess collects within skin and internal organs, interfering with their function

Scleroderma

- Blood vessels and joints can be affected
  - More common in women than in men
  - Difficult to diagnose
    - Only rough estimates to its prevalence
    - Range from 40,000 to 165,000
Scleroderma

- Can be localized or systemic, both groups include subgroups
- Localized
  - Limited to skin and related tissues, and may involve underlying muscle
  - Internal organs not affected
  - Does not progress to systemic forms
  - Conditions often subside, but skin changes and damage from event can be permanent

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Scleroderma

- Two types of localized: morphea and linear
  - Morphea
    - Local patches of hardened skin
    - Red patches of skin develop white centers with purplish borders
    - Lesions remain active for weeks to several years
    - Spontaneous softening that leaves darkened area of skin
    - One or a few patches
Scleroderma

- Two types of localized: morphea and linear
  - Morphea
    - In generalized morphea, patches may grow over large areas of body
    - Can occur at any site
    - Lesions can be extensive and gradually evolve into generalized morphea in which patches extend over most or all of body surface

Scleroderma

- Two types of localized: morphea and linear
  - Linear
    - Single line or band of thickened and discolored skin develops
    - Line usually runs down arm or leg, but sometimes runs down forehead

Scleroderma

- Systemic scleroderma (systemic sclerosis) affects
  - Skin
  - Blood vessels
  - Major organs
Scleroderma

- Two types of systemic disease are recognized: limited and diffuse
  - Limited
    - Skin thickening is generally limited to fingers, forearms, legs, face, and neck
    - Raynaud's phenomenon may be present for years before any other symptoms develop
    - People with this form are less likely than people with diffuse disease to develop severe organ involvement
  - Diffuse
    - Skin thickening may occur anywhere on body, including trunk
    - Only short interval of time will elapse between onset of Raynaud's phenomenon and significant organ involvement
    - Damage typically occurs over first 3 to 5 years, after which most patients enter stable phase that varies in length
    - Organ involvement may lead to diseases and dysfunction of esophagus, GI tract, kidney, heart, and lung

How could Raynaud's phenomenon impact your assessment of a patient who is hypoxic?
Scleroderma Management

• No treatment
• Therapies aimed at preventing or reducing organ damage to prolong life
• Treatment decisions are made by symptom-by-symptom and organ-by organ basis
• Patients with systemic scleroderma can suffer serious organ failure

Scleroderma Management

• Thickening of vessels can lead to
  – Significant hypertension
  – Pulmonary, cardiovascular, and renal failure
• Can present with
  – Hypertensive crisis
  – Renal failure
  – Heart failure

Scleroderma Management

• Drug therapy
  – Nitrates and calcium channel blockers to lower BP
  – ACE inhibitors to reverse renal crisis
• Prehospital care may include both basic and advanced life support
Transplant Complications

- Every 11 minutes, a new name is added to the National Transplant Waiting List
  - Many are waiting for solid organ transplant
    - Kidney
    - Liver
    - Pancreas
    - Heart
    - Lung

- In 2008, 28,000 Americans received organ transplant
  - Likelihood of EMS personnel caring for organ transplant recipient is great
  - Common complications are related to immunosuppression
    - Infection
    - Rejection
    - Drug toxicity

Infection

- Most common life-threatening complication of long-term immunosuppressive therapy
  - Causes
    - Community-acquired bacterial and viral diseases
    - Opportunistic infections
    - Infections due to P. carinii/jirovecii, Nocardia asteroides, Aspergillus spp, Cryptococcus neoformans, CMV, VZV, influenza, respiratory syncytial virus (RSV), R. equi, and Legionella spp
Which signs and symptoms of infection may be masked by immunosuppressive treatment?

Infection

• After infection, inflammatory responses associated with invasion of microbes are impaired by immunosuppressive therapy
  – Results in masking of normal signs and symptoms of infection that would normally be evident
    • Infections are often advanced or have spread at time of patient complaint or clinical presentation
    • Established infection is difficult to treat in immunocompromised transplant recipient
    • Significant antimicrobial toxicities are common, often due to diminished renal or hepatic function and drug interactions

Rejection

• Tissues or cells from another person (except an identical twin) carry nonself markers and may be recognized by body as foreign
  – Why some tissue transplants (grafts) are rejected
• Allografting
  – Transplants donated by nonidentical (genetically unrelated) people
Rejection

• Isografting
  – Transplanted tissue donated by identical twin
• Organ rejection is major complication of solid organ transplant recipients
  – Classified as
    • Hyperacute
    • Acute
    • Chronic

Rejection

• Signs and symptoms of transplant rejection vary, depending on transplanted organ and general health of recipient
  – General signs and symptoms
    • Pain at site of transplant
    • General malaise
    • Irritability (in children)
    • Flu-like symptoms
    • Fever
    • Weight changes
    • Swelling and edema
    • Change in heart rate or BP

Drug Toxicity

• Many drugs are necessary to manage patient with solid organ transplant
  – Most required for life
• Primary goals of drug therapy are to prevent organ rejection and infection
• Major complication of drug therapy is drug toxicity and adverse effects
Drugs Used in Transplants

• Oral immunosuppressant drugs commonly used in the U.S. include interleukin-2 (IL-2) receptor antagonist
  – Daclizumab (Zenapax)
  – Basiliximab (Simulect)
  – OKT3 (monoclonal antibody)

Drugs Used in Transplants

• Other drugs
  – Tacrolimus (Prograf)
  – Mycophenolate mofetil (CellCept)
  – Sirolimus (Rapamune)
  – Prednisone
  – Cyclosporine (Neoral, Sandimmune, Gengraf)
  – Azathioprine (Imuran)

Drugs Used in Transplants

• Medications to help prevent infections
  – Antivirals such as acyclovir (Zovirax), or valganciclovir (Valcyte) to fight viruses
  – Antifungals such as fluconazole (Diflucan), nystatin (Mycostatin, Nilstat), oritraconazole (Sporanox) to fight fungal infections
Drugs Used in Transplants

• Antibiotics used to treat bacterial infections
  – Sulfamethoxazole/trimethoprim (Bactrim, Septra)
• Drugs commonly associated with drug toxicity and adverse drug interactions in transplant patients
  – Cyclosporine
  – Azathioprine
  – Corticosteroids

Transplant Patient Management

• Prehospital care
  – May vary from providing only comfort measures to supporting vital functions
  – Patients often have long and complex medical history
    • Most will require rapid transport

Summary

• Immune system is designed to prevent foreign substances from entering body
  – If that fails, this system should launch attack to find and destroy foreign substances
• Organs of immune system include spleen, tonsils, adenoids, lymph nodes, thymus
Summary

Lymphocytes are primary units of immune system
- B lymphocytes produce antibodies
  - Called humoral immunity
- T lymphocytes provide immune protection with three types of cells
  - Killer T cells attack invading organisms
  - Helper T cells encourage B cell antibody production
  - Suppressor T cells regulate immune response so it does not attack body
- T cells provide cell-mediated immunity

Summary

Natural immunity is present at birth
- Not antigen specific
- Acquired immunity develops after exposure to specific antigens
- Local allergic reactions do not produce life-threatening signs and are treated with antihistamines such as diphenhydramine

Summary

Anaphylaxis is immediate, systemic, and life-threatening reaction
- Antigens are substance that trigger antibody formation
- Antibodies bind to antigen that produced them
  - Antibodies aid in neutralizing antigen and removing it from body
Summary

- Allergic reaction is increased physiological response to an antigen after a previous exposure to same antigen
  - Localized allergic reactions do not affect entire body
- Anaphylaxis is most extreme form of allergic reaction
  - Rapid recognition and aggressive therapy are needed for patient survival

Summary

- Anaphylaxis is form of type I hypersensitivity reaction
- Localized allergic reactions affect skin, nasal passages, or eyes, not lungs or cardiovascular system
  - Are treated with an antihistamine such as diphenhydramine

Summary

- Almost any substance can cause anaphylaxis
  - Risk of anaphylaxis increases with frequency of exposure
- Chemical substances released by basophils and mast cells cause signs and symptoms of anaphylaxis
  - These chemicals include histamine, leukotrienes, and other substances
Summary

• Symptoms of anaphylaxis may include a sudden onset of hives, angioedema, pruritus; sneezing and coughing; airway obstruction; wheezing; hypotension or vascular collapse; chest pain; nausea, vomiting, or diarrhea; and weakness, headache, syncope, seizures, or coma

• Determine whether patient has used an epinephrine autoinjector or taken diphenhydramine prior to arrival

Summary

• Treatment of anaphylaxis includes administration of epinephrine and, if patient is hypotensive, 1 to 2 L of normal saline
  – Additional interventions may include antihistamines, inhaled beta agonists, corticosteroids, glucagon, and vasopressors

• Autoimmune disease occurs when body’s immune system attacks normal body cells, causing harm

Summary

• Collagen vascular disease is also called connective tissue disease

• Systemic lupus erythematosus (SLE), or lupus, is a disease of young women that can damage many organs
  – It often causes a butterfly rash on nose and cheeks
  – Severe damage to GI organs, kidneys, lungs, and CNS are possible
Summary

• Scleroderma means hard skin
  – Caused by increased collagen production
  – Systemic scleroderma may cause Raynaud’s phenomenon and dysfunction of the esophagus, GI tract, kidneys, heart, and lungs
• Solid organs transplanted include kidney, liver, pancreas, heart, and lung

Summary

• Infection, rejection, and drug toxicity are key complications after organ transplant
• Infections may include community-acquired bacterial or viral diseases; opportunistic infections; and others
  – Normal signs and symptoms of infection may be masked by immunosuppressive therapy

Summary

• When body recognizes transplanted tissue as “non-self,” it begins to reject it
  – Rejection may be hyperacute (within minutes), acute (within 1 week), or chronic (more than 1 week)
• Immunosuppressive drugs have many side effects
  – Three drug groups known to cause many adverse effects include cyclosporine, azathioprine, and corticosteroids
Questions?