Nursing Care of Patients Immune Disorders

Chapter 18

Disorders of Immune System

- Three categories
  - Hypersensitivity reactions
    - Anaphylaxis, hemolytic transfusion reactions, measles, transplant rejections
  - Autoimmune disorders
    - Pernicious Anemia, Idiopathic Autoimmune Hemolytic Anemia, Hashimoto's Thyroiditis, Lupus Erythematosus,
    - Ankylosing Spondylitis
  - Immune deficiencies
    - Hypogammaglobulinemia, acquired immunodeficiency syndrome (AIDS)

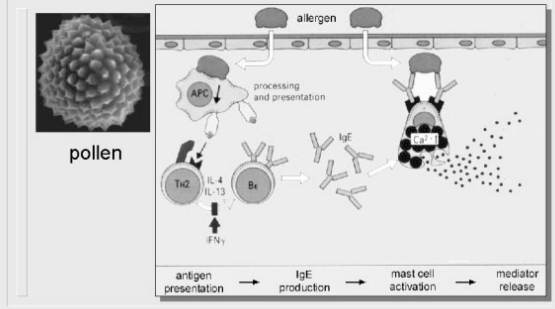
Hypersensitivity Reactions

- Injury to body resulting from its exaggerated response
- Classified by way tissue injured
- Type I, II, III, and IV

Type I

- Anaphylactic reaction
  - Occurs immediately
    - Exposure to specific antigen
  - Mild to severe, life threatening
  - Must have an initial, previous exposure
    - Sensitization
  - Localized or systemic

Sensitization against allergens



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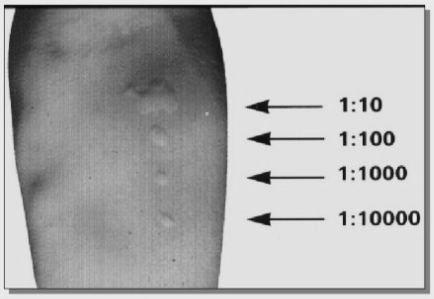
Type I (cont.)

- Severest forms
  - Anaphylaxis
  - Urticaria
  - Angioedema
- Scratch test
  - Done to identify specific allergen
    - Tiny amts of common allergens scratched on skin
      - Observed for s/s of reaction
        - » Redness, edema, pruritis – if occur- considered local rx

Skin test for allergy



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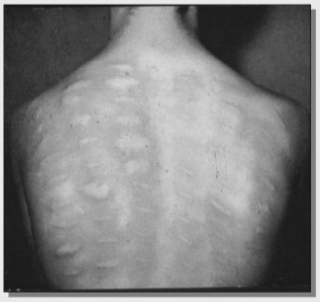
Skin test for allergy



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Type I (cont.)

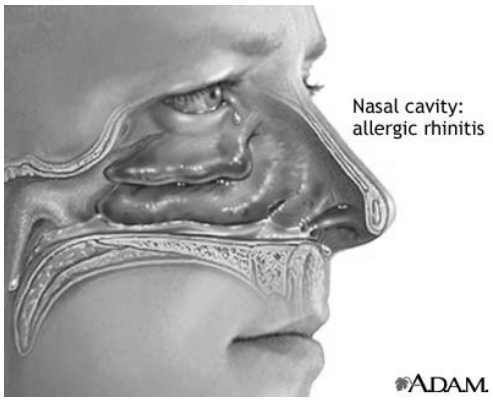
Allergic Rhinitis

- Most common form of allergy
- Perennial
  - s/s throughout year
- Hay fever or seasonal
  - s/s occur seasonally

Type I (cont.)

Allergic Rhinitis

- Pathophysiology
  - Result of an antigen-antibody rx
  - Ciliary action decreases
  - Mucous secretions increase
  - Local tissue edema
    - Vasodilation
- Signs & Symptoms
  - Sneezing, nasal itching, runny nose, itchy red eyes
  - Pale, cyanotic, edematous nasal mucosa
  - Allergic shiners



Type I (cont.)

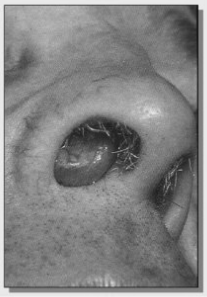
Allergic Rhinitis

- Complications
  - Usually self-limited
  - Sinusitis, nasal polyps, asthma, chronic bronchitis
- Diagnosis
  - Detailed H&P
  - Inspection of nasal passages
  - Skin testing

Many organ are be affected by "allergy"

The nasohprynx

Nasal polyps



Type I (cont.)

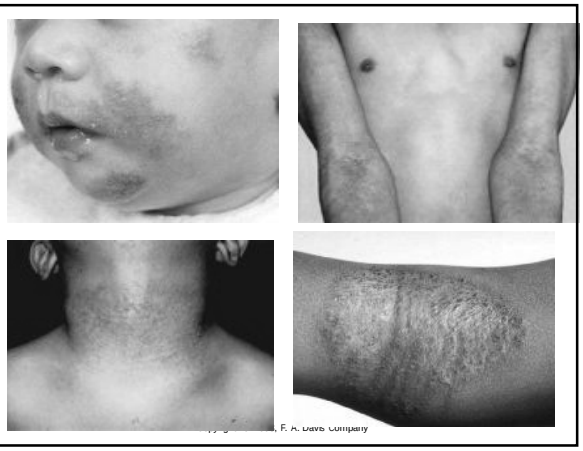
Allergic Rhinitis

- Treatment
  - Elimination of offending allergen
  - Antihistamines/nasal decongestants
  - Corticosteroid sprays if severe s/s
  - Immunotherapy w/debilitating s/s
    - Allergy shots
- Nursing management
  - Education
  - Continued assessment

Type I (cont.)

Atopic Dermatitis

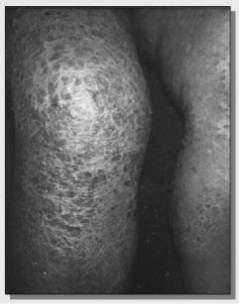
- AKA; Eczema
  - Inflammatory skin response
- Signs & Symptoms
  - Initially
    - Pruritus, edema, extremely dry skin
  - Followed by
    - Blisters that crust over, then become scaled
    - Decreased sweating over these areas
    - Skin becomes thickened



Many organ are be affected by "allergy"

The skin

eczema



Type I (cont.)
Atopic Dermatitis

- Complications
  - Infections
    - Staphylococci
      - Found normally on dermal layer
- Diagnosis
  - No specific tests
  - Detailed H&P

Type I (cont.)
Atopic Dermatitis

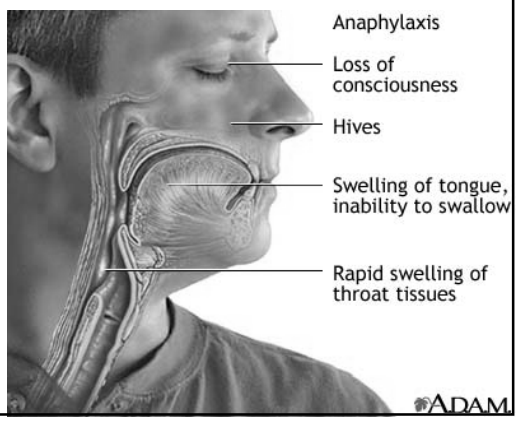
- Treatment
  - Focuses on s/s
    - Oil-in-water lubricants
      - Alpha-Keri oil
    - Topical steroids
    - Topical antibiotics if lesions infected
- Nursing care
  - Assessment and documentation
  - Administer meds
  - Education

Type I (cont.)
Anaphylaxis

- Pathophysiology
  - Severe systemic hypersensitivity reaction
  - Widespread histamine release
- Etiology
  - Numerous (see table 18.1 , pg 303)

Type I (cont.)
Anaphylaxis

- Signs & symptoms
  - Bronchial narrowing: Stridor, wheezing, respiratory arrest
  - Hypotension, tachycardia, cardiac arrest
- Complications
  - Respiratory and cardiac arrest
- Diagnosis
  - No specific
  - H & P
  - Allergy testing after pt stable may be considered



Type I (cont.)
Anaphylaxis

- Immediate treatment guided by symptoms
  - IV access
  - Oxygen
  - Epinephrine
  - Antihistamines
  - Corticosteroids
  - Vasopressors (ie, Dopamine)
  - Mechanical ventilation

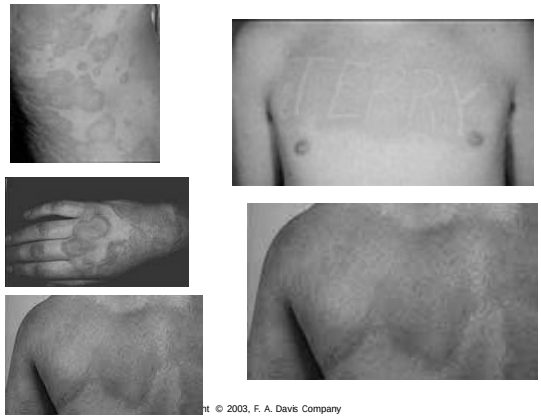
Type I (cont.)
Anaphylaxis

- Nursing care
  - Early recognition and treatment
  - Maintain airway
  - Emotional support
  - Education
    - Avoidance of allergen
    - Anaphylaxis kit or epinephrine pen

http://www.epipen.com/anaphylaxis_main.aspx

Type I (cont.)
Urticaria (Hives)

- Pathophysiology
  - Release of histamine from mast cells
    - Caused by antigen stimulated rx of IgE antibodies
- Etiology
  - Medications, foods, cold, local heat, pressure, stress, others
  - Underlying chronic conditions
    - SLE, lymphoma, hyperthyroidism, cancer, etc.

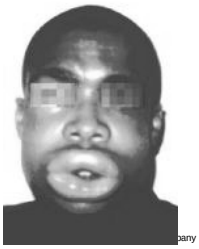


Type I (cont.)
Urticaria (Hives)

- Signs & symptoms
  - Raised, pruritic, nontender, erythematous wheals on skin
  - Usually on trunk and proximal extremities
- Diagnosis
  - H & P
- Treatment
  - Epinephrine
  - Corticosteroids
  - Antihistamines
  - Histamine H_2 blockers
    - Cimetidine or ranitidine

Type I (cont.)
Angioedema

- Pathophysiology
  - Form of urticaria
  - Affects submucosal/subcutaneous tissue rather than skin
- Signs & Symptoms
  - Painless, minimally pruritic
  - Dermal erythematous/subcutaneous eruptions
  - Skin/mucous membrane edema



Type II

- Cell or substance w/antigen attached to cell membrane
  - Destroyed by IgG or IgM
    - Senses as being a foreign antigen
- May be beneficial
  - Bacteria
- May not beneficial
  - RBC sensed as foreign

Type II

Hemolytic Transfusion Reaction

- RBCs with antigens foreign to person rapidly lysed
  - Massive amount of cellular debris
  - Occludes blood vessels throughout body
  - Ischemia, necrosis
  - Life threatening

Type II

Hemolytic Transfusion Reaction

- Etiology
  - Prior sensitization
    - Bacterial or viral infection – occasionally
    - Prior blood transfusion or past pregnancy
      - Most common
      - Maternal and fetal Rh factors are different
      - Mother becomes sensitized by the fetal Rh type
      - Affects future fetuses

Type II

Hemolytic Transfusion Reaction

- Signs & Symptoms
  - Sudden onset of low back/chest pain
  - Hypotension
  - Fever, chills
  - Tachycardia, tachypnea, wheezing, dyspnea
  - Urticaria
  - Anxiety, headache, nausea

Type II

Hemolytic Transfusion Reaction

- Diagnosis
  - Direct Coombs' test
- Prevention
  - Rhogam: Rh_0 (D) negative patients
  - Careful blood transfusion administration

Type II

Hemolytic Transfusion Reaction

- Treatment
  - – Depends on severity/organs affected
  - – Antihistamines
  - – Corticosteroids
  - – Epinephrine
  - – Diuretics
    - Assist with excretion of cellular debris
      - – Prevent occlusion of kidneys

Type III

- Immune complexes formed by antigens and antibodies
  - – Usually of the IgG type
  - – Pt already sensitized with an initial antigen exposure
  - – Reaction occurs on subsequent exposure
- Antigen-antibody complexes form within blood vessels
  - – Lead to blood vessel damage
    - Red, edematous skin lesion, hemorrhage, necrosis

Type III

Serum Sickness

- Antigen-antibody complexes cause symptoms of inflammation
- 7 to 10 days after penicillin/sulfonamide
- Signs & Symptoms
  - – Severe urticaria and angioedema
- Usually brief and self-limiting condition
  - – Can become chronic

Type III

Serum Sickness

- Diagnosis
  - – ↑ WBC & ESR, ↓ complement assay
- Medical treatment
  - – Antipyretics
  - – Antihistamines
  - – Epinephrine
  - – Corticosteroids – if severe

Type IV

- Delayed reaction
- Sensitized T lymphocyte contacts antigen to which its sensitized
- Cell-mediated immune response
- Necrosis

Type IV

Contact Dermatitis

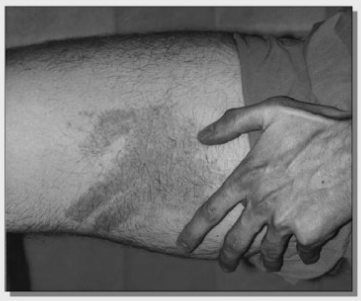
- Pathophysiology
  - – Chemical comes in contact with skin
  - – On second exposure, T cells secrete chemicals
- Most common etiologic factors
  - – Poison ivy, poison oak, latex rubber

Type IV

Contact Dermatitis

- Signs & symptoms
  - – Reddened, pruritic, fragile vesicles
    - Onset usually within hours of exposure
- Diagnosis
  - – Appearance of skin lesions/history
- Treatment
  - – Symptom control
    - Oral/topical antihistamines or corticosteroids

Contact dermatitis reaction to mango sap



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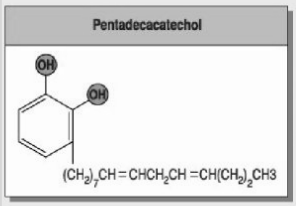
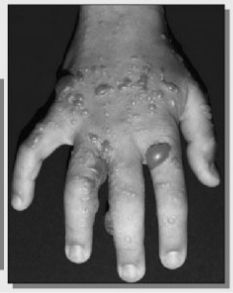
Poison ivy / poison oak reaction

active hapten molecule





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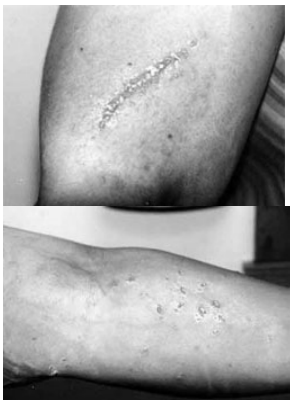
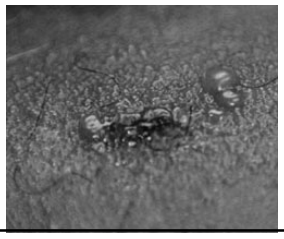
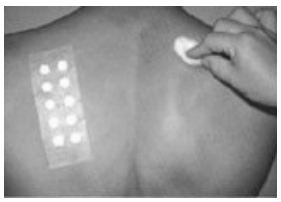






Type IV
Transplant Rejection

- Transplanted living tissue sensed as foreign
- Lymphocytes sensitized immediately after transplant
- Invade transplanted tissue and destroy it
- S/S reflect failure of specific organ or tissue
- Prevention
  - Immunosuppression therapy
- Infection can result in death

Autoimmune Disorders

- Immune system recognizes body's own cell antigens as foreign
- Immune response destroys them
- Influencing factors
  - Viral infections
  - Drugs
  - Cross-reactive antibodies

Autoimmune Disorders — Pernicious Anemia

- Pathophysiology
  - Antibodies against gastric parietal cells and intrinsic factor
  - Vitamin B_{12} deficiency
  - RBC production decreased
- Etiology
  - Familial tendency
  - Gastric or small bowel resections
    - If vitamin B12 or intrinsic factor not replaced

Autoimmune Disorders — Pernicious Anemia

- Diagnosis
  - Macrocytic (large) RBCs
  - Low vitamin B12 levels
  - Schilling test
  - Gastric secretion analysis
    - Measures HCL levels
      - Will be low or absent
- Treatment
  - Corticosteroids
  - Lifelong vitamin B_{12} IM
    - Initially weekly – then monthly

Autoimmune Disorders — Pernicious Anemia

- Nursing management
  - Administer vitamin B12 as prescribed
  - Ambulation w/frequent rest periods
  - Assistance w/ADLs
  - Education
    - Lifelong treatment and follow-up

Autoimmune Disorders
Idiopathic Autoimmune Hemolytic Anemia

- Pathophysiology
  - Autoantibodies attach to RBCs
    - Causes lysis or agglutination (clumping)
      - Lysis - Fragments of destroyed RBCs circulate in blood
      - Agglutination – causes occlusion and ischemia
- Signs & symptoms
  - Vary to mild to severe
  - Fatigue, pallor, hypotension, dyspnea, palpitations, jaundice

Autoimmune Disorders
Idiopathic Autoimmune Hemolytic Anemia

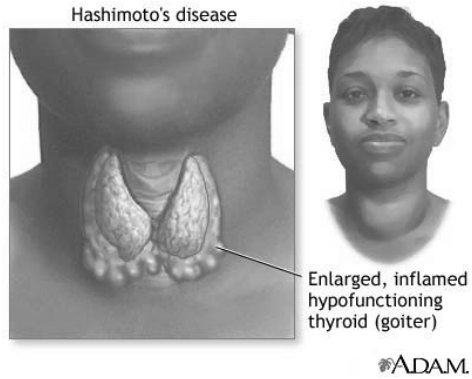
- Diagnosis
  - ↓ RBC, Hgb & Hct
  - Fragmented RBCs on microscopic exam
  - ↑ LDH
    - Due to destroyed RBCs and ischemia
- Treatment
  - Immunosuppressive medications & corticosteroids
  - Oxygen
  - Folic acid
  - Blood transfusions & erythrocytapheresis
  - Splenectomy

Autoimmune Disorders
Hashimoto's Thyroiditis

- Pathophysiology
  - Auto antibodies for TSH
    - Causes thyroid gland over stimulation
      - Thyromegaly
        - » Due to overstimulation, infiltration w/lympocytes and phagocytes → causes inflammation and further enlargement
  - Auto antibodies destroy thyroid
    - Hypothyroidism

Autoimmune Disorders
Hashimoto's Thyroiditis

- Etiology
  - Unknown
  - More common
    - Women by 8 times
    - 30 – 50 years old
    - Down syndrome and Turner's syndrome
- Signs & symptoms
  - Initially same as hyperthyroidism
  - Then progress to hypothyroidism
  - Goiter



Autoimmune Disorders
Hashimoto's Thyroiditis

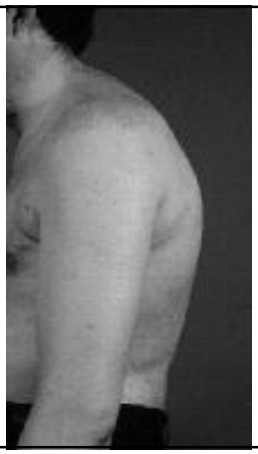
- Diagnosis
  - Immunofluorescent assay
  - ↑ TSH, ↓ T3 & T4
  - Thyroid scan
- Treatment
  - Lifelong thyroxine replacement therapy

Autoimmune Disorders
Ankylosing Spondylitis

- Pathophysiology
  - Chronic progressive inflammatory disease of sacroiliac, costovertebral, large peripheral joints
- Etiology
  - Familial tendency
  - Affects men more frequently

Autoimmune Disorders — Ankylosing Spondylitis

- Signs & symptoms
  - Lower back stiffness, pain, lordosis, kyphosis, spasms, fatigue, anorexia, weight loss
- Treatment
  - No cure, supportive care, surgery
- Education: ROM frequently



Immune Deficiencies

- One or more components of immune system completely absent or deficient
  - Inability to elicit or sustain an adequate immune response
  - Unable to fight off an infectious agent

Immune Deficiencies — Hypogammaglobulinemia

- Pathophysiology
  - Absence or deficiency of one or more of 5 immunoglobulins from defective B cell function
  - Prone to infections
- Etiology
  - Hereditary congenital disorder
    - More common in males
    - Usually have a normal lifespan
  - Acquired after childhood from unknown cause

Immune Deficiencies — Hypogammaglobulinemia

- Signs & symptoms
  - Asymptomatic until 6 months
    - When maternal immunoglobulins are gone
  - Recurrent infections
    - Especially staph & strep
- Treatment
  - Minimizing infections
  - IgG injections
  - Fresh frozen plasma replaces IgM
  - IgA cannot be replaced
    - Increases risk of pulmonary infections

Lupus Erythematosus

- Three types
  - Discoid (DLE)
    - Skin lesions only
  - Drug-induced systemic lupus erythematosus
    - After certain medications
    - Table 18.8 Page 314

Lupus Erythematosus

- Systemic lupus erythematosus
  - Chronic, inflammatory, multisystem disorder
  - Body develops antibodies against self
  - Very unpredictable
  - Flare triggers
    - Table 18.9 Page 316

Etiology

- Common in women 20 to 40
- Higher incidence in African American and Hispanic women
- Familial tendency

Signs and Symptoms

- Vary from mild to severe
- Any system in the body can be affected with symptoms related to organ involved
  - Musculoskeletal—arthralgia, arthritis
  - Hematologic—anemia, leukocytopenia, thrombocytopenia, elevated ESR, false + VDRL
  - Cardiopulmonary—pericarditis, myocarditis, MI, pleurisy, valvular heart disease

Signs and Symptoms

- Renal—renal failure, UTI, fluid and electrolyte imbalances
- Central Nervous System—cranial neuropathies, cognitive impairment, mental changes, seizures
- Gastrointestinal—anorexia, ascites, pancreatitis
- Ophthalmologic—conjunctivitis, dry eyes, glaucoma, cataracts, retinal pigmentation

Diagnostic Testing

- ANA (antinuclear antibody)
  - Measures autoantibodies
- Anti-Sm
  - Specific immunoglobulin for SLE
- Anti-nDNA
  - + in 60 to 80% of SLE patients
  - ESR and C-reactive protein are non-specific

Therapeutic Interventions

- Education
  - Made aware of flare triggers and how to avoid
  - Planning to avoid fatigue
  - Minimum 8 hours sleep, plan naps during day
  - Exercise program very important
  - Warmth and cool compress therapy
  - Well-balanced diet

Therapeutic Interventions

- Medical alert bracelet
- Encourage smokers to quit
- Alert to s/s of bleeding, cardiac, or renal problems
- Support groups
- Allow patient to express emotions
- Lupus Foundation of America