Topics

- Type III hypersensitivity
- Type IV hypersensitivity

18-1

Type III hypersensitivity

- Immune complex – mediated
- Activates complement
- Inflammation

18-2

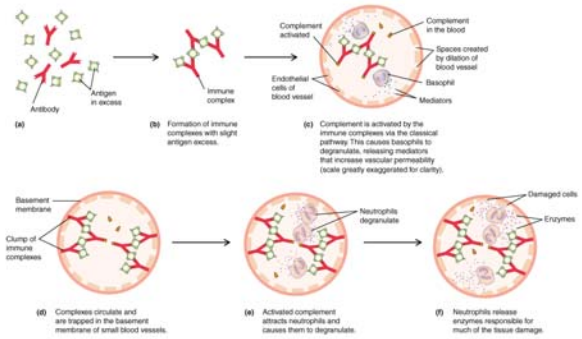
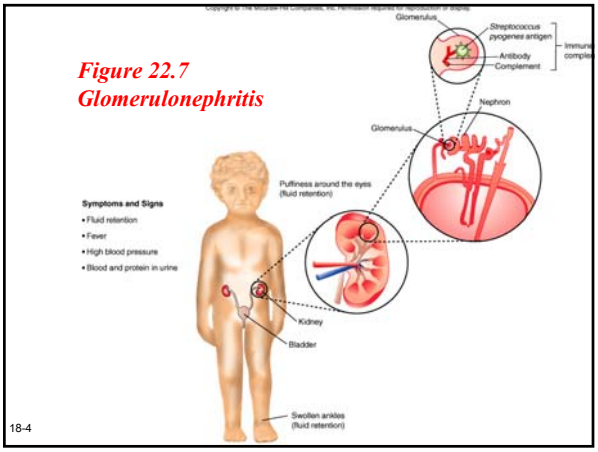


Figure 18.5 Immune complex - mediated

18-3



18-4

TABLE 18.3 Pathogenesis of Immune Complex Disease

1. Antibody combines with excess soluble antigen.
2. The antibody-antigen combination reacts with complement.
3. Complexes are deposited in sites such as skin, kidney, and joints.
4. Fragments of complement cause release of histamine and other mediator substances from mast cells or basophils and also attract neutrophils.
5. Release of the mediators causes increased permeability of blood vessel walls.
6. Immune complexes penetrate or form in blood vessel walls.
7. Neutrophils enter the vessel walls chemotactically.
8. Neutrophils release lysosomal enzymes, especially proteases, that induce tissue injury.

18-5

Type III hypersensitivity and disease

Excess antigen
farmer's lung
bacterial endocarditis
malaria
Streptococcus pyogenes skin and throat infections
acute glomerulonephritis
Rubella (German measles), early symptoms
rash, painful joints, fever
Disseminated intravascular coagulation
clots form in small blood vessels; organ failure
Arthus reaction – local reaction in response to injected antigen
Serum sickness – passive immunization with animal serum
antisera against diphtheria, tetanus
Antigens in foreign serum induce immune response

18-6

Type IV hypersensitivity

- Delayed cell – mediated
- Tuberculin skin test
- Contact hypersensitivities *(contact dermatitis)*

18-7

Type IV hypersensitivity

- Delayed cell – mediated
- Tuberculin skin test
- Contact hypersensitivities
- Infectious diseases

18-8

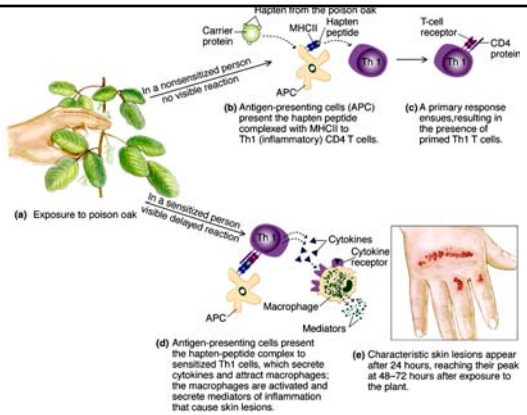
Delayed cell - mediated

- Delayed hypersensitivity
- Sensitized T lymphocytes

18-9

18-10

Figure 18.6 Tuberculin skin test



18-11

Figure 18.7 Contact hypersensitivities

Figure 18.8 Severe contact hypersensitivity

18-12

Common examples of contact hypersensitivity

poison ivy, poison oak
nickel in metal jewelry
chromium salts in leather products
cosmetics
latex products (IgE-mediated Type I reactions also)
plant protein induces sensitization
use vinyl gloves
potential allergens detected with patch test

18-13

Infectious disease

- **Protective function cause tissue damage**
- **Ex. Leprosy** *(damaged sensory nerves; Mycobacterium leprae)*
- **Tuberculosis** *(granulomas form – tubercules;*
  persistent Mycobacterium tuberculosis infection)
- **Leishmaniasis** *(Leishmania species survive within macrophages)*
- **Herpes simplex** *(HSV-1, HSV-2 infects nerve cells*
  emerges as cold sores, genital herpes)

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TABLE 18.1 Some Characteristics of the Major Types of Hypersensitivities

Characteristic	Type I hypersensitivity Immediate; IgE-mediated	Type II hypersensitivity Cytotoxic	Type III hypersensitivity Immune complex–mediated	Type IV hypersensitivity Delayed cell-mediated
Cell type responsible	B cells	B cells	B cells	T cells
Type of antigen	Soluble	Cell-bound	Soluble	Soluble or cell-bound
Type of antibody	IgE	IgG, IgM	IgG	None
Other cells involved	Basophils, mast cells	Red blood cells, white blood cells, platelets	Various host cells	Various host cells
Mediators	Histamine, serotonin, leukotrienes	Complement, ADCC	Complement, neutrophil proteases	Cytokines
Transfer of hypersensitivity	By serum	By serum	By serum	By T cells
Time of reaction after challenge with antigen	Immediate, up to 30 minutes	Hours to days	Hours to days	Peaks at 48 to 72 hours
Skin reaction	Wheal and flare	None	Arthus	Induration, necrosis
Examples	Anaphylactic shock, hay fever, hives	Transfusion reaction, hemolytic disease of newborns	Serum sickness, farmer's lung, malarial kidney damage	Tuberculin reaction, contact dermatitis, tissue transplant rejection

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