Chapter 10: Alterations in Immune Function
Test Bank

MULTIPLE CHOICE

1. Dramatic hypotension sometimes accompanies type I hypersensitivity reactions, because
   a. massive histamine release from mast cells leads to vasodilation.
   b. toxins released into the blood interfere with cardiac function.
   c. anaphylaxis results in large volume losses secondary to sweating.
   d. hypoxia due to bronchoconstriction impairs cardiac function.

   ANS: A
   Hypotension can occur in type I hypersensitivity due to massive histamine release leading to vasodilation. Toxins are not released during type I hypersensitivity reactions. Sweating occurs as a reaction to shock from severe hypotension; the hypotension occurs first and is due to histamine release. Hypoxia occurs in anaphylaxis due to shock from severe hypotension; the hypotension occurs first and is due to histamine release.

   REF: Pgs. 199-200

2. Which disorder is associated with a type III hypersensitivity mechanism of injury?
   a. Systemic lupus erythematosus
   b. Graves disease
   c. Erythroblastosis fetalis
   d. Seasonal allergic rhinitis

   ANS: A
   Systemic lupus erythematosus is a type III hypersensitivity disorder. Type III hypersensitivity is characterized by antigen-antibody complex deposition into tissues, with consequent activation of complement and a subsequent self-sustaining inflammatory reaction. Graves disease and erythroblastosis fetalis are type II hypersensitivity reactions. Seasonal allergic rhinitis is a type I hypersensitivity reaction.

   REF: Pgs. 204-206

3. A child with a history of recent strep throat infection develops glomerulonephritis. This is most likely a type _____ hypersensitivity reaction.
   a. I
   b. II
   c. III
   d. IV

   ANS: C
   Immune complex glomerulonephritis (an inflammatory renal disorder) is an example of a type III hypersensitivity reaction. The circulating immune complex is then deposited in the glomerular capillary wall and mesangium. Glomerulonephritis secondary to strep throat is not a type I, II, or IV sensitivity reaction.

   REF: Pg. 206

4. RhoGAM (an Rh antibody) would be appropriate in an Rh-_____ woman with an _____ Rh-_____ antibody titer carrying an Rh-_____ fetus.
   a. negative; positive; positive
   b. positive; negative; negative
   c. negative; negative; positive
   d. negative; negative; negative

   ANS: C
   If a woman is Rh-negative, RhoGAM is administered for prevention of Rh-positive antibodies. Erythroblastosis fetalis develops during pregnancy when an Rh-negative mother is sensitized to her fetus's Rh-positive red cell group antigens because of exposure during her current or a previous pregnancy. RhoGAM contains antibodies against Rh antigens on fetal blood cells and is given to the mother to destroy fetal cells that may be present in her circulation before her immune system becomes activated and begins to produce anti-Rh antibodies. RhoGAM is not effective if the mother already has a positive antibody titer for fetal Rh antigens. An Rh-positive woman with negative Rh antibody titer carrying Rh-negative fetus does not require RhoGAM because the mother is Rh-positive and the fetus is Rh-negative.

   REF: Pg. 203

5. Which disorder is considered a primary immunodeficiency disease?
   a. HIV/AIDS
   b. Malnutrition immunodeficiency
   c. Cancer immunodeficiency
   d. Radiation immunodeficiency

   ANS: A
   HIV/AIDS is a primary immunodeficiency disease involving destruction of T helper cells. Malnutrition immunodeficiency is a secondary immunodeficiency disorder and leads to T-cell destruction and dysfunction. Cancer immunodeficiency is a secondary immunodeficiency disorder caused by the destruction of rapidly proliferating cells from chemotherapeutic agents. Radiation immunodeficiency is a secondary immunodeficiency disorder caused by the destruction of rapidly proliferating cells from the effects of radiation.

   REF: Pg. 209

6. Patients with immunodeficiency disorders are usually first identified because they
   a. run high fevers.
   b. have unusually high WBC counts.
   c. develop brain infections.
   d. develop recurrent infections.

   ANS: D
   The first clinical indicators of immunodeficiency disorders are the signs and symptoms of infection, and the disorders are often first suspected when an individual has severe recurrent, unusual, or unmanageable infections. High fevers can occur in patients who have an intact immune system. Because of the immune deficiency, patients with immunodeficiency disorders may not demonstrate expected WBC counts with infection. Infections in patients with immunodeficiency disorders can occur anywhere in the body.

REF: Pg. 209

7. Severe combined immunodeficiency (SCID) syndrome is an example of a(n)
   a. deficient immune response.
   b. excessive immune response.
   c. primary acquired immunodeficiency.
   d. hypersensitivity reaction.

   ANS: A
   SCID syndrome is an example of a deficient immune response in which the immune response is ineffective due to disease-causing genotypes or secondary/acquired dysfunction. An excessive immune response includes disorders in which the immune system is overfunctioning or hyperfunctioning. HIV/AIDS is an example of a primary acquired immunodeficiency disorder. Hypersensitivity reactions are an excessive immune response.

   REF: Pg. 209

8. Certain autoimmune diseases are associated with the presence of specific proteins on a person's cells. These proteins are called _____ proteins.
   a. complement
   b. antibody receptor
   c. HLA or MHC
   d. TCR or BCR

   ANS: C
   Human major histocompatibility complex (MHC) genes located on chromosome 6p21 (also called human leukocyte antigen (HLA) genes) are frequently associated with certain autoimmune disorders. Complement, antibody receptors, and TCR or BCR are not factors in development of autoimmune disease.

   REF: Pg. 197

9. The most common primary immune deficiency that affects only B cells is
   a. DiGeorge.
   b. Bruton agammaglobulinemia.
   c. Wiskott-Aldrich.
   d. selective IgA.

   ANS: D
   The most common B-cell primary immunodeficiency disorder is selective IgA deficiency. This disorder affects 1:2000 persons. DiGeorge is a T cell primary immune deficiency. Bruton agammaglobulinemia is not the most common primary immune deficiency affecting B cells; frequency of disease is 1:250,000 males. Females are carriers. Wiskott-Aldrich affects both T cells and B cells.

   REF: Pg. 211

10. Transfusion reactions involve RBC destruction caused by
    a. donor antigens.
    b. recipient antibodies.
    c. donor T cells.

d. recipient T cells.

ANS: B
The recipient of the blood transfusion has antibodies to the donor's red blood cell (RBC) antigens; the antibodies destroy large numbers of RBC. Donor antigens, donor T, and recipient T cells do not cause transfusion reactions.

REF: Pgs. 201-202

11. The hypersensitivity reaction that does not involve antibody production is type
    a. I.
    b. II.
    c. III.
    d. IV.

    ANS: D
    Type IV hypersensitivity reactions do not involve antibody production. The principal mediators are lymphocytes, including T helper cells (Th) that mediate the reaction by releasing lymphokines (cytokines) and/or antigen-sensitized cytotoxic T cells (Tc) that can directly kill cells. The other types involve antibody production. Type I involves IgE; type II involves IgM or IgG; type III involves IgG.

    REF: Pg. 206

12. The principle Ig mediator of type I hypersensitivity reactions is
    a. IgA.
    b. IgG.
    c. IgM.
    d. IgE.

    ANS: D
    Immunoglobulin E (IgE) is the principal antibody mediating type I hypersensitivity reactions. IgA is not the primary Ig mediator of type I hypersensitivity reactions. IgG is involved in type II hypersensitivity reactions. IgM is involved in type II hypersensitivity reactions.

    REF: Pg. 199

13. Myasthenia gravis is a type II hypersensitivity disorder that involves
    a. impaired muscle function.
    b. symptoms of hyperthyroidism.
    c. symptoms of arthritis or polyarthralgia.
    d. symptoms of glomerular disease.

    ANS: A
    Myasthenia gravis involves muscle weakness due to loss of acetylcholine stimulation at the motor end plate. Symptoms of hyperthyroidism occur in Graves disease. Symptoms of arthritis or polyarthralgia occur in systemic lupus erythematosus, a type II hypersensitivity disorder. Glomerular disease can occur in type III hypersensitivity disorders.

    REF: Pg. 203

14. A primary effector cell of the type I hypersensitivity response is

a. monocytes.
b. mast cells.
c. neutrophils.
d. cytotoxic cells.

ANS: B
Mast cells are a primary effector cell of the type I hypersensitivity response. Monocytes, neutrophils, and cytotoxic cells are not primary effectors of the type I hypersensitivity response.

REF: Pg. 199

15. An important mediator of a type I hypersensitivity reaction is
a. complement.
b. antigen-antibody immune complexes.
c. T cells.
d. histamine.

ANS: D
Histamine mediates type I hypersensitivity reactions. Complement mediates type II hypersensitivity reactions. Antigen-antibody immune complexes mediate type III hypersensitivity reactions. T cells mediate type IV hypersensitivity reactions.

REF: Pg. 199

16. The effects of histamine release include
a. vasoconstriction.
b. bronchodilation.
c. increased vascular permeability.
d. decreased gut permeability.

ANS: C
Histamine release leads to increased vascular permeability, which fosters fluid movement out of capillaries and into tissues leading to the edema common in type I hypersensitivity. Histamine leads to bronchoconstriction, increased gut permeability, and vasodilation (not vasoconstriction).

REF: Pgs. 199-200

MULTIPLE RESPONSE

17. Autoimmune diseases result from *(Select all that apply.)*
a. overactive immune function.
b. increase in self-tolerance.
c. failure of the immune system to differentiate self and nonself molecules.
d. communicable infections.

ANS: A, C

Autoimmunity results from an functional increase in the activity of the immune system. Breakdown of self-tolerance occurs. Genetic and environmental factors are thought to contribute to autoimmunity. Autoimmune disease results from failure of the immune system to differentiate self and nonself molecules; this results in immune reactions that attack normal tissue.

REF: Pg. 196

18. Secondary immune deficiency problems may be caused by *(Select all that apply.)*
    a. surgery.
    b. high blood sugar.
    c. corticosteroids.
    d. genetic disorders.
    e. low protein level.

    ANS: A, B, C, E
    Surgery leads to reduced B and T cell counts. High blood sugar decreases WBC function. Corticosteroids are immunosuppressive. Antibodies are composed of proteins; low protein level impairs antibody synthesis. Genetic abnormalities of immune components lead to primary immune deficiency problems.

    REF: Pg. 212

19. Anaphylaxis may occur in certain hypersensitivity reactions, including type *(Select all that apply.)*
    a. I.
    b. II.
    c. III.
    d. IV.

    ANS: A, B
    Anaphylaxis can occur in type I and type II hypersensitivity reactions. Anaphylaxis does not occur in type III or type IV reactions.

    REF: Pg. 200 | Pg. 202

COMPLETION

20. Seasonal allergic rhinitis is most involved in type ____ hypersensitivity reactions.

    ANS:
    I
    Seasonal allergic rhinitis is most involved in type I hypersensitivity reactions.

    REF: Pgs. 199-200