Exam 1: NSG530/ NSG 530 (Latest 2023/ 2024) - Advanced Pathophysiology Exam | Questions and Verified Answers| 100% Correct| Grade A

Q: When antibodies are formed against red blood cell antigens of the Rh system, how are the blood cells destroyed?
a. Complement-mediated cell lysis b. Phagocytosis by macrophages c. Phagocytosis in the spleen d. Neutrophil granules and toxic oxygen products

Answer:
C
Antibodies against platelet-specific antigens or against red blood cell antigens of the Rh system coat those cells at low density, resulting in their preferential removal by phagocytosis in the spleen, rather than by complement-mediated lysis. These blood cells are not destroyed by complement-mediated cell lysis, phagocytosis by macrophages, neutrophil granules, or toxic oxygen products.

Q: When soluble antigens from infectious agents enter circulation, what is tissue damage a result of?
a. Complement-mediated cell lysis b. Phagocytosis by macrophages c. Phagocytosis in the spleen d. Neutrophil granules and toxic oxygen products

Answer:
D
Of the options available, only the components of neutrophil granules as well as the several toxic oxygen products produced by these cells, damage the tissue.

Q: How are target cells destroyed in a type II hypersensitivity reaction?
a. Complement-mediated cell lysis b. Phagocytosis by macrophages
c. Neutrophil granules and toxic oxygen products d. Natural killer cells

Answer:
D

The mechanism that results in a type II hypersensitivity reaction involves a sub- population of cytotoxic cells that are not antigen specific (natural killer [NK] cells). Antibody on the target cell is recognized by Fc receptors on the NK cells, which releases toxic substances that destroy the target cell. The other options do not cause the destruction of target cells related to a type II hypersensitivity reaction.

Q: Graves disease (hyperthyroidism) is an example of which type II hyper- sensitivity reaction?
a. Modulation
b. Antibody-dependent cell-mediated cytotoxicity c. Neutrophil-mediated damage
d. Complement-mediated lysis

Answer:
A
The antibody reacts with the receptors on the target cell surface and modulates the function of the receptor by preventing interactions with their normal ligands, replacing the ligand and inappropriately stimulating the receptor or destroying the receptor. For example, in the hyperthyroidism (excessive thyroid activity) of Graves disease, autoantibody binds to and activates receptors for thyroid-stimulating hor- mone (TSH) (a pituitary hormone that controls the production of the hormone thyroxine by the thyroid). Graves disease is not a result of cell-mediated cytotoxicity, neutrophil-mediated damage, or complement-mediated lysis.

Q: Type III hypersensitivity reactions are a result of which of these?
a. Antibodies coating mast cells by binding to receptors that signal its degran- ulation, followed by the discharge of preformed mediators
b. Antibodies binding to soluble antigens that were released into body fluids and the immune complexes being deposited in the tissues
c. Tc cells or lymphokine-producing Th1 cells directly attacking and destroying cellular targets
d. Antibodies binding to the antigen on the cell surface

Answer:
B
Antigen-antibody (immune) complexes that are formed in the circulation and then deposited later in vessel walls or extravascular tissues cause most type III hypersen- sitivity diseases. Type III hypersensitivity reactions are not the result of antibodies coating mast cells to signal their degranulation, immune cells directly attacking and destroying targets, or antibodies binding to the antigen on the cell surface.

Q: A type IV hypersensitivity reaction causes which result?
a. Antibodies coating mast cells by binding to receptors that signal its degran- ulation, followed by the discharge of preformed mediators
b. Antibodies binding to soluble antigens that were released into body fluids and the immune complexes being deposited in the tissues
c. Lymphokine-producing Th1 cells directly attacking and destroying cellular targets
d. Antibodies binding to the antigen on the cell surface

Answer:
C
Type I, II, and III hypersensitivity reactions are mediated by antibody, type IV reactions are mediated by T lymphocytes and do not involve antibody. Type IV mechanisms occur through either Tc cells or lymphokine-producing Th1 cells. Tc cells directly attack and destroy cellular targets.

Q: In a type III hypersensitivity reaction, the harmful effects after the immune complexes that are deposited in tissues are a result of what?
a. Cytotoxic T cells
b. Natural killer cells
c. Complement activation
d. Degranulation of mast cells

Answer:
C
Complement activation, particularly through the generation of chemotactic factors for neutrophils, causes the harmful effects of immune complex deposition. The neutrophils bind to antibody and C3b contained in the complexes and attempt to ingest the immune complexes. Type III hypersensitivity reactions as described are not the result of cytotoxic T cells, natural killer cells, or degranulation of mast cells.

Q: Raynaud phenomenon is classified as a type III hypersensitivity reaction and is due to:
a. Immune complexes that are deposited in capillary beds, blocking circulation b. Mast cells that are bound to specific endothelial receptors, causing them to degranulate and creating a localized inflammatory reaction that occludes capillary
circulation
c. Cytotoxic T cells that attack and destroy the capillaries so that they are unable to perfuse local tissues
d. Antibodies that detect the capillaries as foreign protein and destroy them using lysosomal enzymes and toxic oxygen species