MCQ IMMUNOLOGY

1) In atopy the common allergens are all except:

1- Antitetanic serum
2- Penicillin
3- Pollens
4- Streptomycin
5- House mice
6- House dust

2) Initial response of type I hypersensitivity reaction is characterized by:

1- Vasodilatation
2- Vascular leakage
3- Smooth muscle spasm
4- All of the above
5- None of the above

3) The commonest manifestations of atopy are:

1- Hay fever
2- Extrinsic asthma
3- Atopic eczema in infancy & childhood
4- All of the above
5- None of the above

4) The commonest manifestations of atopy are all except:

1- Hay fever
2- Extrinsic asthma
3- Heart failure
4- Atopic eczema in infancy & childhood

5) Anaphylaxis shock results in:

1- Urticaria
2- Generalized edema
3- Bronchospasm
4- Wide spread vasodilatation & circulatory failure.
5- All of the above
6- None of the above

6) Anaphylaxis shock results in all except:

1- Urticaria
2- Generalized edema
3- Systemic hypertension
4- Bronchospasm
5- Vasodilatation & circulatory failure.

7) Antibody mechanisms involved in type II hypersensitivity reaction are:

1. Complement dependent reaction.
2. Antibody dependent cell mediated cytotoxisity (ADCC)
3. Antibody mediated cellular dysfunction
4. **All of the above**
5. None of the above

8) Antibody mechanisms involved in type II hypersensitivity reaction are all except:

1. Complement dependent reaction.
2. **Anaphylactic shock**
3. Antibody dependent cell mediated cytotoxisity (ADCC)
4. Antibody mediated cellular dysfunction

9) Clinically type II hypersensitivity reaction occurs in the following situations:

1. Transfusion reactions.
2. Erythroblastosis fetalis.
3. Autoimmune hemolytic anemia.
4. Autoimmune thrombocytopenic purpura.
5. Good Pasteur's Syndrome.
6. **All of the above**
7. None of the above

10) Clinically type II hypersensitivity reaction in occurs in the following situations except:

1. Transfusion reactions.
2. Autoimmune thrombocytopenic purpura.
3. Agranylocytosis.
4. Good Pasteur's Syndrome.
5. **Acute serum sickness**
6. Erythroblastosis fetalis.
7. Autoimmune hemolytic anemia.

11) Antibody dependent cell mediated cytotoxisity may be relevant to:

1. Destruction of targets too large to be phagocytosed e.g: parasites
2. Destruction of tumor cells
3. Play a role in graft rejection.
4. **All of the above**
5. None of the above

12) Antibody mediated cellular dysfunction occurs in:

1. Myasthenia gravis
2. Graves' disease
3. **Both of the above**
4. None of the above

13) Antibody mediated cellular dysfunction occurs in all except:

1. Myasthenia gravis
2. Graves' disease
3. **Cardiomyopathy**

14) Arthus reaction is:

1. A localized type III reaction
2. It occurs at the site of injection of soluble antigens
3. It depends on the presence of precipitating antibodies in the circulation
4. Ag/Ab complexes are formed in & around post-capillary venules
5. The inflammatory reaction develops over 4-8 hours.
6. **All of the above**
7. None of the above

15) Immune complexes disease is characterized by:

1. Formation of Ag/Ab complexes in the circulation.
2. Deposition of ICs in various tissues
3. Initiating an inflammatory reaction throughout the body.
4. **All of the above**
5. None of the above

16) The resulting pathologic lesions of immun-complex deposition are:

1. Vasculitis
2. Glomerulonephritis
3. Arthritis
4. **All of the above**
5. None of the above

17) The resulting pathologic lesions of immun-complex deposition are all except:

1. Vasculitis if it occurs in blood vessels
2. **Good Pasteur's Syndrome if it occurs in renal glomeruli and lung alveoli.**
3. Glomerulonephritis if it occurs in the renal glomeruli
4. Arthritis if it occurs in joints

18) The resulting pathologic lesions of immun-complex deposition are all except:

1. Vasculitis if it occurs in blood vessels.
2. **Graves' disease if it occurs in thyroid acini.**
3. Glomerulonephritis if it occurs in the renal glomeruli
4. Arthritis if it occurs in joints

19) Clinical features of immune complex deposition are:

1. Fever
2. Urticaria
3. Arthralgia
4. Lymph node enlargement
5. Proteinurea
6. **All of the above**
7. None of the above

20) Clinical features of immune complex deposition are all except:

1. Fever
2. Urticaria
3. **Myopathy**
4. Arthralgia
5. Lymph node enlargement
6. Proteinurea

21) Type IV hypersensitivity:

1. Produces tissue injury independent on the production of antibody.
2. Can be transferred to a non sensitized individual by using lymphocytes from a sensitized donor.
3. Inflammatory lesions develop slowly.
4. Important in host defense against infection by intracellular organisms.
5. **All of the above**
6. None of the above

22) Features of type IV hypersensitivity are all except:

1. Produces tissue injury independent on the production of antibody.
2. Can be transferred to a non sensitized individual by using lymphocytes from a sensitized donor.
3. **Inflammatory lesions develop rapidly.**
4. Important in host defense against infection by intracellular organisms.

23) Features of type IV hypersensitivity are all except:

1. **Produces tissue injury dependent on the production of antibody.**
2. Can be transferred to a non sensitized individual by using lymphocytes from a sensitized donor.
3. Inflammatory lesions develop slowly.
4. Important in host defense against infection by intracellular organisms.

24) Examples of delayed type hypersensitivity reaction are all except:

1. Tuberculin test
2. Contact dermatitis
3. **Rheumatoid artheritis**

25) In contact dermatitis the induction of skin sensitivity may occur:

1. Seven days of the initial exposure.
2. Over months or years of repeated exposure to small amounts of antigen
3. **Both of the above**
4. None of the above

26) T-cell mediated cytotoxisity play an important role in:

1. Graft rejection
2. Resistance against viral infection.
3. **Both of the above**
4. None of the above

27) T-cell mediated cytotoxisity play an important role in all except:

1. Graft rejection
2. **Resistance against pyogenic bacteria.**
3. Resistance against viral infection.

28) Cytotoxic T cells kill their targets by:

1. Drilling holes into the membrane → water to inter the cell causing osmotic lysis.
2. Delivering proteases into the target cells → activation of apoptosis (PCD).
3. **Both of the above**
4. None of the above

29) What is true in hyperacute graft rejection:

1. Occurs within minutes or few hours after transplantation.
2. Damage appears in the vessel walls with vascular thrombosis & graft necrosis.
3. The rejection reaction depends on the presence of preformed humoral antibodies.
4. **All of the above.**
5. None of the above

30) What is true in acute graft rejection?

1. Occurs within days to few weeks after transplantation.
2. The graft shows edema, hemorrhage, thrombosis and finally necrosis.
3. Both humoral & cell mediated mechanisms are involved.
4. Occurs early in untreated patients.
5. Associated with vasculitis, and interstitial mononuclear cell infiltrate.
6. **All of the above.**
7. None of the above

31) Chronic rejection is characterized by:

1. Gradually occurs
2. Cell mediated immune mechanism is responsible.
3. Occurs in patients whose graft performed under immuno-suppression.
4. Graft vessels become obstructed by interstitial fibrosis & infiltrating lymphocytes.
5. **All of the above.**
6. None of the above

32) Mechanisms preventing anti-self reactivity in healthy individuals are:

1. Clonal deletion
2. Clonal anergy
3. Peripheral suppression of T cells
4. **All of the above.**
5. None of the above

33) Loss of self tolerance may be due to:

1. By-pass of helper T cell tolerance
2. Polyclonal lymphocytic activation
3. Imbalance between suppressor and helper T cell function
4. Emergence of sequestered antigens
5. **All of the above.**
6. None of the above

34) Genetic factors play a significant role in the predisposition to autoimmune diseases as evidenced by:

1. Familial clustering of autoimmune diseases as SLE, autoimmune hemolytic anemia & autoimmune thyroiditis
2. Linkage of several autoimmune diseases with HLA antigens: e.g; Type I diabetes & HLA DR 3 & 4.
3. Introduction of human HLA B27 gene in transegenic mice → disease similar to ankylosing spondylitis in man.
4. **All of the above**
5. None of the above

35) Autoimmune diseases include all except:

1. Single organ or specific diseases in which the immune response is directed against one particular organ or cell type.
2. Multi-system diseases characterized by lesions in many organs & associated with multiplicity of auto-antibodies or cell mediated reaction or both.
3. **Two system affection e.g., lung and kidney in Good Pasteur's Syndrome.**

36) Single organ specific autoimmune diseases include:

1. Hashimoto's thyroiditis
2. Graves' disease
3. Autoimmune hemolytic anemia
4. Autoimmune orchitis
5. Atrophic gastritis & pernicious anemia
6. **All of the above**
7. None of the above

37) Single organ specific autoimmune diseases include all except:

1. Hashimoto's thyroiditis
2. Graves' disease
3. **Rheumatoid artheritis**
4. Autoimmune hemolytic anemia
5. Autoimmune orchitis

38) Systemic autoimmune diseases include:

1. Systemic lupus erythermatosis
2. Rheumatoid artheritis
3. Sjogern's syndrome
4. Reiter's syndrome
5. **All of the above**
6. None of the above

39) Systemic autoimmune diseases include all except:

1. Systemic lupus erythermatosis
2. Rheumatoid arthritis
3. **Graves' disease**
4. Reiter's syndrome

40) Primary immunodeficiency diseases include:

1. X- linked agamaglobulinemia (Bruton's disease)
2. Isolated deficiency of Ig A
3. Thymic hypoplasia (De George syndrome)
4. Genetic deficiencies of complement system
5. **All of the above**
6. None of the above

41) Primary immunodeficiency diseases include all except:

1. X- linked agamaglobulinemia (Bruton's disease)
2. Isolated deficiency of Ig A
3. **Sjogern's syndrome**
4. Thymic hypoplasia (De George syndrome)

42) X-linked agamaglobulinemia (Bruton's disease) is characterized by:

1. Failure of B-cells to differentiate into plasma cells.
2. X linked disease.
3. Becomes apparent around 6 months of age.
4. Manifested by recurrent pharyngitis, sinusitis, otitis media, bronchitis & pneumonia.
5. **All of the above**
6. None of the above

43) Isolated deficiency of IgA is characterized by:

1. Is the most common primary immunodeficiency disease.
2. Mostly asymptomatic.
3. Weakened mucosal defenses predispose patients to recurrent upper respiratory tract infection, diarrhea & autoimmune diseases.
4. **All of the above**
5. None of the above

45) Thymic hypoplasia (De George syndrome) is characterized by:

1. Results from a lack of thymic influence on the immune system.
2. Thymus is usually rudimentary & T cells are deficient or absent.
3. Infant is extremely vulnerable to viral, fungal & protozoal infections.
4. Is due to congenital malformation affecting the third & fourth branchial pouches.
5. Sometimes associated with parathyroid hypoplasia.
6. **All of the above**
7. None of the above

46) Thymic hypoplasia (De George syndrome) is characterized by all except:

1. Results from a lack of thymic influence on the immune system.
2. **Thymus is usually rudimentary & T cells are deficient or absent.**
3. Infant is extremely vulnerable to viral, fungal & protozoal infections.
4. Is due to congenital malformation affecting the third & fourth branchial pouches.
5. Sometimes associated with parathyroid hypoplasia.

47) Genetic deficiencies of complement system immunodeficiency diseases are characterized by:

1. Hereditary deficiency of C3 serious recurrent pyogenic infection.
2. Inherited deficiency of C1, C2 & C4 → increased susceptibility to immune complex diseases as SLE.
3. Deficiencies of C5-C8 → recurrent neisserial infections; gonococcal & meningeococcal.
4. **All of the above**
5. None of the above

48) Secondary immunodeficiency diseases are characterized by:

1. Encountered in patients with malnutrition, infection, cancer, renal diseases.
2. Occur in patients receiving immuno-suppressive drugs and/or corticosteroids.
3. Commoner than primary immunodeficiency diseases.
4. The most famous example is the "Acquired Immunodeficiency Syndrome; AIDs".
5. **All of the above**
6. None of the above

49) Secondary immunodeficiency diseases are characterized by all except:

1. Encountered in patients with malnutrition, infection, cancer, renal diseases.
2. Occur in patients receiving immuno-suppressive drugs and/or corticosteroids.
3. **They are less common than primary immunodeficiency diseases.**
4. The most famous example is the "Acquired Immunodeficiency Syndrome or AIDs".
5. All of the above.

50) Secondary immunodeficiency diseases are characterized by all except:

1. Encountered in patients with malnutrition, infection, cancer, renal diseases.
2. Occur in patients receiving immuno-suppressive drugs and/or corticosteroids.
3. Commoner than primary immunodeficiency diseases.
4. **The most famous example is Thymic hypoplasia (De George syndrome).**

True or False:

1. Hypersensitivity reaction is an exaggerated response by an individual to an antigen, following a previous exposure.
2. Hypersensitivity reaction consists of an exaggerated response by an individual to an antigen, following **a first exposure**.
3. Type I hypersensitivity reactions is an immediate one
4. **Type IV** hypersensitivity reactions is an **immediate** one
5. **Type II** hypersensitivity reactions is a **delayed** one

6. Anaphylaxis is a rapidly occurring reaction that follows the combination of an antigen & antibodies previously bound to the surface of the mast cells or basophiles.
7. **Atopic** diseases are **a localized type III** hypersensitivity reaction.
8. Anaphylaxis shock is a systemic type I hypersensitivity reaction.
9. Sufferer from hay fever develops acute inflammation of nasal & conjunctival mucosae with sneezing, ↑ nasal & lacrimal secretion **within days** of exposure to gross pollens.
10. An attack of asthma with difficult wheezy respiration due to narrowing of the airways by bronchospasm & ↑ mucous secretion develops rapidly when the asthmatic patients inhale allergens.
11. Anaphylaxis shock results from injection of antitetanic sera or penicillin in **non** sensitized individual.
12. The targets of cytotoxic antibodies in type II hypersensitivity are the cells of the blood (R.B.Cs, W.B.Cs & platelets).
13. The antigens in type II HSR may be intrinsic to the cell membrane.
14. In Good Pasteur's Syndrome anti-basement membrane antibodies are formed against antigens **in both liver** & alveolar capillaries.
15. In antibody dependent cell mediated cytotoxicity cell lyses occurs without phagocytosis.
16. Antibody dependent cell mediated cytotoxisity may be relevant to destruction of targets too large to be phagocytosed (e.g: parasites & tumor cells).
17. In antibody mediated cellular dysfunction, antibodies directed against cell surface receptors impair or deregulate function without causing cell injury or inflammation.
18. In myasthenia gravis: antibodies react with **adrenaline receptors** on the motor end plates of the skeletal muscle → weakness.
19. In Type III HSR immune complexes are formed by union between antibodies and antigens either locally in the tissues or in the circulation.
20. Arthus reaction is defined as a localized area of tissue necrosis resulting from acute immune complexe vasculitis usually elicited in the skin.
21. Arthus reaction is defined as a **generalized tissue** necrosis in the skin resulting from acute immune complexe vasculitis.
22. Low levels of circulating immune complex occur transiently in normal individual without producing tissue injury.
23. If immune complexes are deposited in the glomerular capillaries, they cause tissue damage; **type III-HSR**.
24. Immune complexes formed with "marked antigen excess" are small, soluble & don't activate the complement, so they are not harmful.

25. Immune complexes formed in “slight antigen excess”, in “slight antibody excess” are insoluble → activate complement, so it is harmful.
26. Large immune complexes formed in great antibody excess are rapidly removed from the circulation by the mononuclear phagocytic cells, so are relatively harmless.
27. Immune complexes leave the circulation and become deposited on the basement membrane in renal glomeruli and synovial membranes as their endothelium is fenestrated.
28. The deposition of immune complexes in the blood vessels which are lined by continuous endothelium e.g: skin and endocardium, depends on increased capillary permeability.
29. Once immune complex deposited in the tissues they initiate an acute inflammatory reaction approximately **10 minutes** after antigen administration.
30. Whatever the immune complex deposited, the tissue reaction is similar.
31. Type IV HSR can be transferred to a non sensitized individual by using **lymphocytes** from a sensitized donor.
32. In type IV-HSR the inflammatory lesions develop **rapidly**.
33. Type IV HSR is important in host defense against infection by intracellular organisms such as, viruses, certain bacteria, fungi & protozoa.
34. Tuberculin test is produced by intra-cutaneous injection of a protein lipopolysacharide component of the tubercle bacilli in a previously sensitized individual.
35. Tuberculin test is produced by intra-cutaneous injection of **tubercle bacilli** in a previously sensitized individual.
36. In tuberculin test the main cause of indurations is ↑ vascular permeability → dermal edema & interstitial deposition of fibrin.
37. Epithelioid cells are **lymphocytes** undergo morphologic transformation into epithelial like cells.
38. Granuloma is a microscopic aggregation of epithelioid cells, surrounded by a collar of lymphocytes and this pattern of inflammation is called granulomatous inflammation.
39. In contact dermatitis the induction of skin sensitivity occurs over months or years of repeated exposure to small amounts of antigen.
40. For a graft to be accepted; it must be antigenically compatible with the tissue of the host.
41. The greater the differences in the antigenic barrier, the greater the likelihood of graft rejection.
42. The hyperacute rejection reaction depends on the presence of preformed humoral antibodies that react immediately with the graft.

43. Acute graft rejection represents a primary response; where both humoral & cell mediated immune mechanisms are involved.
44. Chronic rejection reaction depends on cell mediated immunity.
45. Rejection of allogenic bone marrow graft is mediated by the relatively radiation resistant T cells & NK cells.
46. Self tolerance is due to **proliferation** of self reactive clones of T cells, B cells or both during their maturation.
47. Immune tolerance is defined as a state in which the individual is incapable of developing an immune response against specific antigens.
48. Self tolerance refers to a lack of immune response to the individuals own tissue antigens.
49. Self tolerance refers **to an immune response** to the individuals **own tissue antigens**.
50. Clonal deletion is loss or deletion of self reactive clones of **plasma cells** during their maturation.
51. In clonal deletion T cells bearing receptors for self antigens are detected within the thymus and deleted by apoptosis.
52. Clonal deletion affects mainly self reactive T cells, but plays a less important role in B cell tolerance.
53. Clonal deletion occurs mainly in the **spleen**.
54. Clonal anergy is a prolonged or irreversible functional inactivation of lymphocytes induced by encounter with antigen under certain conditions.
55. Colonal anergy is a state of inactivation of lymphocytes occurs if the antigen presenting cells don't express co-stimulators.
56. Peripheral suppression of T cells is defined as inactivation of both T-cells & B-cells mediated by suppressor T-cells.
57. Peripheral suppression of T cells is defined as **activation** of both T-cells & B-cells mediated by **helper** T-cells.
58. Breakdown of one or more of self tolerance leads to the development of **immunodeficiency diseases**.
59. Antibody response occurs only when potentially self-reactive B-cells receive help from T-cells.
60. In rheumatic heart disease, which follows infection with streptococcal antigens an immune response against such microbes may produce a tissue damaging reaction by recognizing & damaging the cardiac valves.
61. Self tolerance is maintained by anergy of autoreactive lymphocytes that were not deleted during development.
62. Autoimmunity may occur if self reactive but anergic lymphocytes are stimulated & reactivated by antigen independent mechanisms.

63. Endotoxins may act as powerful stimulant → activation of all B cells including self reactive anergic lymphocytes.
64. Sympathetic ophthalmitis is an immunologically mediated inflammation of both eyes following trauma to one eye → an immune response against sequestered ocular antigens → immunologically mediated damage of the target antigens in both eyes.
65. Clinically, immunodeficiency diseases present with increased susceptibility to infections & sometimes cancer.
66. Patients with defects in immunoglobulin components typically suffer from recurrent pyogenic infections.
67. Patients having defects in **cell mediated** immunity are prone to infections by **pyogenic** bacteria.
68. Primary immunodeficiency diseases come to attention in early life because of the increased vulnerability of the child to viral infections.
69. Acquired Immunodeficiency Syndrome or AIDs is characterized ↑ liability of opportunistic infections, secondary neoplasms & neurological manifestation.
70. Secondary IDDs are commoner than primary IDDs.

Thank you