Inflammation I

1**.Classsic signs of inflammation seem to be the following:**

1.pain
2.local heat
3.disurbances in function
4.swelling
5.fever
6.redness

2.**Pain as a symptom of inflammation can be provoked by**:

1.epinephrine
2.acidosis
3.hyperkalemia
4.bradykinin
5.PgE-2
6.sodium accumulation

3.The following factors can induce pain in the site of inflammation:

1.PgE-2
2.H+ and K+ hyperionia
3.P substance
4..epinephrine
5.local heat
6.swelling

4.The following factors could predispose tissue to active hyperemia:

1.high hyaluronidase activity
2.mediators of inflammation
3.low tone of vasodilators
4.norepinephrine
5.axon-reflex
6.accumulation of acid metabolites

5.The following assertions are true:

1.axon –reflex is realized due to participation of nociceptive endings of nociceptive fibres which belong to group C
2.axon-reflex needs CNS participation to realize itself
3.axon-reflex is realized due to participation of nervus sympathcus terminates
4. axon-reflex takes part in active hyperemia formation

6.The signs of inflammatory active hyperemia are:

1increasing in linear blood velocity
2.exudation
3.low capillary hydrostatic pressure
4.increased content of oxyhemoglobin in veinous blood
5.increased number of plasma capillaries

7.Peculiarity of active inflammatory hyperemia are the following:

1.part of capillaries doesn't work
2.maximal dilation of capillaries is characteristic
3.all microcirculatory vessels are involved in the process
4.blood shunting is characteristic

8.The signs of congestion in inflammation obviously the following:

1.dilation of microcirculatory vessels
2.slowdown of blood flow

3.low blood vessels sensitivity to tonic influences
4.decreasing in capillary hydrostatic pressure
5.increased permeability of capillaries and postcapillary veins

9.The following factors predispose to inflammatory stasis formation:

1.dilation of supplying artery
2.exudate formation
3.spasm of precapillary sphincters
4.erythrocytes hemolysis
5.slowdown of blood flow
6.low blood viscosity

10.Mediators of inflammation originated from plasma are the following:

1.PAF(platelet activating factor)
2.opsonins
3.lysozomal enzymes
4.bradykinin
5.leukotriens
6.the products of fibrinolysis

11.Mediators of inflammation of cellular origin are:

1.PAF (platelet activating factor)
2.histamine
3.clotting factors
4.serotonin
5. bradykinin
6.activated complement components

12.Aspirin via blocking cyclooxygenase inhibits formation of:

1.PgE-2
2.TXA-2 (thromboxane)
3.PgI-2(prostacyclin)
4.SRS-a (slow reacting substance of anaphylaxis)
5. PAF (platelet activating factor
6.LTD-4

13.Preformed mediators of an acute inflammation are obviously the following:

1.serotonin
2.bradykinin
3.histamine
4.PAF
5.leukotriens
6.prostaglandins

14.Phospholipase A-2 blockers inhibit the following substances formation:

1.arachidonic acid
2.C5a complement component
3.leukotriens B-4 and D-4
4.TXA-2 (thromboxane)
5.histamine
6.PAF (platelet activating factor)

15.The following substances are the derivates of arachidonic acid according to cyclooxygenase pathway of its metabolism:

1.TXA-2 (thromboxane)
2.leukotriens
3.PgE-2
4.PgI-2 (prostacyclin)
5.MRS-a (slow reacting substance of anaphylaxis)

16.The following biological active substances are responsible for increased vessels permeability in course of acute inflammation:

1.heparin
2.histamine
3.bradykinin
4.epynephrine
5.serotonin

17. Membrane phospholipids obviously are the source of the following inflammatory mediators:

1.bradykinin
2.PAF (platelet activating factor)
3.prostaglandins
4.anaphylatoxins
5.leukotriens

: **18. Membrane phospholipids seem to be a source of the following mediators of inflammation**

1.bradykinin
2.anaphylatoxins
3.PAF (platelet activating factor)
4.prostaglandins
5.leukotriens

19.Enzymatic convertion of arachidonic acid may result in formation of:

1.leukotrien B-4
2.C3b complement component
3.thromboxane A-2
4.PAF(platelet activating factor)
5.prostacyclin

20.Characteristic features of histamine are :

1.mast cell granules are its storage
2.monovalent chematractant for neutrophils
3.monovalent chemattractant for eosinophils
4.strong spasmogen for smooth muscle
5.responsible for prolong vessels permeability

21.Histamine possesses by the following characteristics:

1.excites the nociceptive endings of nociceptive fibres
2.responsible for the late phase of increased vessels permeability
3.dilates microcirculatory vessels
4.belongs to preformed mediators of acute inflammation
5.basophils contain it like mast cells

22.The following mediatiors are originated from cell membrane phospholipids:

1.leukotrien B-4
2.bradykinin
3.PgD2
4.PAF (platelet activating factor)
5. thromboxane A-2

23.The products of cyclooxygenase pathway of arachidonic acid seem to be the following substances:

1.PAF (platelet activating factor
2.prostaglandin D-2
3. prostacyclin
4. MRS-a (slow reacting substance of anaphylaxis
5.thromboxane A-2

24.The products of complement system activation can provoke:

1.chemotaxis of polymorphonuclear leukocytes
2.sensitization to painful stimules of nociceptors
3.attack and lysis of the cells
4.liberation of histamine from mast cells
5.faciliating of phagocytosis

25. Characteristic features of C5 complement component are:

1.of phospholipid chemical structure
2.provokes mast cell degranulation
3.named anaphylatoxin
4.opsonin
5. chemattractant

26.In man serotonin posesses by the following characteristics:

1. provokes spasm of veinules
2. mostly originated from platelets
3.dilates arterioles
4.originated from mast cells
5. strong chemattractant for neutrophils

27.To kallikrein-kinin system belong:

1.bradykinin
2.complement components
3.prekallikrein
4.Hageman's factor
5.alpha-2 plasma globulins

28.Activated Hageman's factor initiates:

1.complement system activation
2.splitting of kininogen up to bradykinin formation
3.activation of factor XI of clotting cascade
4.splitting and activation of kallikrein
5.plasmin system activation

29.Characteristic features of bradykinin are:

1.factor Hageman's starts initiates its formation
2.it is preformed mediator
3.arises from membrane phospholipids
4.originated from plasma protein
5.increases blood vessels permeability
6. provokes pain

30.The source of vasoactive kinins seem to be the following:

1.phospholipids of cell membrane
2. alpha-2 plasma proteins
3. mast cells
4.platelets
5.fibrin

31.Start of kallikrein-kinin system activation is possible after a contact of plasma with s

the following surfaces

1.urea acid crystals
2.intact endothelial cells
3.vessels basal membrane
4.bacterial lipopolysaccharides

32.Bradykinin actions in acute inflammation seem to be the following:

1.increases microcirculatory vessels permeability

2. provokes spasm of smooth muscle
3. decreases arterial pressure
4.provokes pain
5.stimulates chemotaxis of neutrophils

33.Activation of kallikrein-kinin system starts with:
1.Hageman's factor activation
2. assembly of C5b6789 complex
3.bradykinin synthesizing
4.conversion of prekallikrein into kallikrein

34.**Endothelial cells produce:**
1.PgD-2
2.PgE-2
3.histamine
4.NO
5.PgI-2

35. The following immunoglobulins interaction with specific antigen can activate complement system:
1.IgA, 2. IgG, 3.IgE, 4.IgM

36. Possible functions of activated complement components in inflammation are the following:
1.chemattractive for phagocytes
2.opsoninization of phagocytes
3. mast cell degranulation
4. cell lysis
5. contraction of smooth muscles

37.The products of arachidonic acid metabolism according to lipooxygenase pathway are:
1.thromboxane A-2
2.leukotriens
3.prostaglandins
4.PAF (platelet activating factor)
5.PgI-2 (prostacyclin)

38.The sources of inflammatory mediators seem to be the following:
1.erythrocytes
2.platelets
3.endothelial cells
4.basophils
5.macrophages

39. **Acute inflammatory response characteristic of:**
1.increased vessels permeability
2.granulomas formation
3.prevalence of neutrophils in the site of inflammation
4.accumulation of polynuclear gigantic cells
5.exudate formation

40.**Chronic inflammation characteristic of:**
1.prevalence of exudation in the site of inflammation
2.appearance of polynuclear gigantic cells
3.accumulation of the neutrophils

4.prevalence of mononuclear cells in the site of inflammation
5.granulomas formation

41.Accumulation of eosinophils in exudate is an evidence of the following possible etiologic factors action:

1.allergy
2.helmints
3 burns
4.radiation
5.protozoa

42.The following physical and chemical parameters characterize the site of an acute

Inflammation:

1.alkalosis
2.hypernatremia
3.high colloid-osmotic pressure
4.hyperkalemia
5.low oncotic pressure