

## Chapter 2. Acute and chronic inflammation(6):

1. In acute inflammation, which events occur in the correct chronological order?  
(Remembered from 2000, 2004 exam.) p50
  - (a) transient vasoconstriction, stasis of blood flow, increased permeability, vasodilation
  - (b) transient vasoconstriction, increased permeability, vasodilation, stasis of blood flow
  - (c) vasodilation, increased permeability, stasis of blood flow, neutrophil accumulation
  - (d) neutrophil accumulation, vasodilation, increased permeability, stasis of blood flow
  - (e) transient vasoconstriction, vasodilation, stasis of blood flow, endothelial gap formation in venules.
2. The first vascular response to injury is
  - (a) slowing of the circulation
  - (b) venular dilatation
  - (c) recruitment of the vascular beds
  - (d) capillary enlargement
  - (e) arteriolar vasoconstriction
3. The first event in acute inflammation is (2000, 2006)
  - (a) arteriolar vasodilation
  - (b) increased permeability
  - (c) diapedesis
  - (d) arteriolar vasoconstriction
  - (e) stasis
4. Leukocytes move into the tissues from the vasculature (extravasation)
  - (a) by the action of actin and myosin
  - (b) predominantly as monocytes on the first day post injury
  - (c) in response to C3b
  - (d) in response to the Fc fragment of IgG
  - (e) largely in the arterioles
5. Regarding the chemical mediators of inflammation
  - (a) histamine is derived from plasma
  - (b) C3b is within macrophages
  - (c) the kinin system is activated in platelets
  - (d) nitric oxide is preformed in leukocytes
  - (e) Serotonin is preformed in mast cells
6. Regarding the complement cascade which of the following statements is true?  
(2004, 2006)
  - (a) the alternative pathway is stimulated by antigen-antibody interaction
  - (b) C5a is split to C5b.
  - (c) C5a activates the lipooxygenase pathway of arachidonic acid metabolism in neutrophils.
  - (d) C3bBb inhibits the final common pathway
  - (e) Microbial surfaces initiate the classical pathway of the complement cascade.

7. Regarding the kinin cascade (2004)

- (a) Kallikrein feeds back to activate factor XI, and amplifies the clotting cascade
- (b) activates the complement cascade via the lectin pathway
- (c) Kallikrein directly cleaves fibrin
- (d) Bradykinin is formed from kallikrein acting on HMWK
- (e) Bradykinin amplifies its own production by cleaving prekallikrein to active kallikrein

8. Bradykinin (2006)

- (a) causes smooth muscle dilatation
- (b) kallikrein causes prohormone degradation to produce bradykinin
- (c) is inhibited by Hageman factor
- (d) ameliorates pain
- (e) is a potent vasoconstrictor
- (x) is factor 12

9. Which of the following is involved in the initiation of the clotting cascade, complement and kinin systems? (2004)

- (a) Hageman factor (XII)
- (b) factor VII
- (c) antigen-antibody complex
- (d) Tissue factor
- (e) Platelet activating factor

10. Which of the following immune cell is unable to phagocytose

- (a) neutrophils
- (b) eosinophils
- (c) macrophages
- (d) T-cells
- (e) monocytes

11. The most common peripheral circulating lymphocyte is the (2006)

- (a) B-cell
- (b) T-cell
- (c) Macrophage
- (d) Natural killer cell
- (e) Polymorphic nucleocyte

12. Macrophages are derived from

- (a) monocytes
- (b) T cells
- (c) B cells
- (d) Eosinophils
- (e) Plasma cells

13. Macrophages may secrete

- (a) histamine
- (b) serotonin
- (c) prostaglandins
- (d) oxygen free radicals
- (e) none of the above

14. Regarding Chronic inflammation, which is correct? (2006)

- (a) it characterised by hyperaemia, oedema, and leukocyte infiltration
- (b) monocytes use the same chemotactic pathways as neutrophils
- (c) it is always preceded by acute inflammation
- (d) most frequently results in resolution
- (e) angiogenesis is not a feature

15. Regarding chronic inflammation, which is correct?

- (a) Macrophages have a half-life of 5 days
- (b) it is always preceded by acute inflammation
- (c) unlike acute inflammation, lymphocytes do not play a major role
- (d) prolonged exposure to toxins such as silica causes repeated bouts of acute inflammation, rather than the chronic type
- (e) attempts at healing are evident

16. Regarding chronic inflammation which of the following is FALSE?

- (a) It is associated with persistent infections
- (b) It primarily involves tissue destruction
- (c) It may contribute to the formation of atherosclerosis
- (d) It can be caused by exposure to toxic agents
- (e) It involves mononuclear inflammatory cells

## Answers

**note (\*) denotes made up question to complete the whole format**

1. In acute inflammation, which events occur in the correct chronological order? p50.

**(c) (transient vasoconstriction), vasodilation, increased permeability, stasis of blood flow, neutrophil accumulation.**

2. The first vascular response to injury is p50

(a) slowing of the circulation (4<sup>th</sup>)

(b) venular dilatation (2<sup>nd</sup>)

(c) recruitment of the vascular beds: *not in the text*

(d) capillary enlargement 2<sup>nd</sup>ary to arteriolar dilation ∴ 2<sup>nd</sup> event

**(e) arteriolar vasoconstriction (1<sup>st</sup>)**

3. The first event in inflammation is p50 (2000 MCQ)

(a) vasodilation 2<sup>nd</sup>

(b) increased permeability 3<sup>rd</sup>

(c) diapedesis (*later with leukocyte involvement*)

**(d) vasoconstriction 1<sup>st</sup>**

(e) increased viscosity (4<sup>th</sup>), stasis (5<sup>th</sup>)

4. Leukocytes move into the tissues from the vasculature (extravasation) (fig 2-8 p57)

**(a) by the action of actin and myosin, in association with a number of actin-regulating proteins**

(b) predominantly as *neutrophils* on the first day post injury, monocytes predominate on day two

(c) in response to chemical gradients (chemotaxis) *endogenous: C5a, lipooxygenase pathway LTB<sub>4</sub>, IL-8, exogenous: bacterial products* and the actions of integrins (leukocytes) and selectins (endothelial cells)

(d) in response to the Fc fragment of IgG, C3 complement, cause *activation*, and phagocytosis, *NOT* extravasation

(e) largely in the *venules*

5. Regarding the chemical mediators of inflammation p64, 65, 127

(a) histamine is widely distributed in tissues and is a *biogenic amine* derived from *basophils and mast cells*

(b) C3b is an activated complement fragment, and is present in the plasma (p81)

(c) Platelets contain α granules contain fibrinogen, fibronectin, factor V, VIII, PDGF, TGF-β; δ granules contain ATP, ADP, serotonin, histamine, and adrenaline, and *do not have a role in the kinin system.*

(d) nitric oxide is released on *activation of leukocytes, by increases in intracellular calcium*

**(e) Serotonin is preformed in platelets and the mast cells of rodents, not of humans**

6. (From 2004 paper and previous other papers) p65-66

Regarding the complement cascade which of the following statements is true?

(a) Antigen-antibody complexes initiate the complement cascade through the *classical* pathway

(b) C5 is split to C5a and C5b.

(c) **C5a activates the lipooxygenase pathway of arachidonic acid metabolism in neutrophils.**

(d) C3bBb (known as C3 convertase) *splits C3 to C3b.*

(e) Microbial surfaces initiate the alternative pathway of the complement cascade.

7. Regarding the kinin cascade p66-67 (2004)

(a) Kallikrein feeds back to activate factor **XII**, and amplifies the clotting cascade

(b) activates the complement cascade by cleaving C3 to C3a and C3b

(c) Kallikrein activates *plasminogen to plasmin, which cleaves fibrin*

(d) **Bradykinin is formed from kallikrein acting on HMWK (HMWK has factor XII activating action too)**

(e) *kinins acts in a similar way to histamine.* They are primarily tissue hormones. They contract visceral smooth muscle, but relax vascular smooth muscle via NO, lowering BP. There is no evidence of a positive feedback loop

8. Bradykinin (p65-66)

(a) causes smooth muscle *contraction, but interestingly, vasodilation!??*

(b) **kallikrein causes prohormone degradation (of HMWK) to produce bradykinin**

(c) in produced by *Hageman factor, which activates prekallikrein to kallikrein, which then converts HMWK to bradykinin*

\*(d) *causes pain* when injected into the skin

\*(e) is a potent *vasodilator*

9. Which of the following is involved in the initiation of the clotting cascade, complement and kinin systems? (2004) (p67, 1278)

(a) **Hageman factor (XII): activates prekallikrein, kallikrein activates plasminogen to plasmin, which can then change C3 to C3a. XII activates the intrinsic clotting pathway.**

(b) factor VII (*extrinsic pathway*)

(c) antigen-antibody complex *alternative pathway of complement only*

\*(d) Tissue factor (*extrinsic pathway*)

\*(e) Platelet activating factor (*stimulates broncho/vasodilation*)

10. Which of the following immune cell is unable to phagocytose

(a) neutrophils

(b) eosinophils

(c) macrophages

(d) **T-cells**

(e) monocytes, natural killer cells.

11. The most common peripheral circulating lymphocyte is the

(a) B-cell (**25%**)

(b) **T-cell: 75% in the peripheral circulation**

(c) macrophage (*not a lymphocyte*)

(d) Natural killer cell (*not a lymphocyte*)

(e) polymorphic nucleocyte (*not a lymphocyte*)

12. Macrophages are derived from p621

(a) **monocytes, which are non-activated, and circulating, whereas monocytes in tissues are macrophages**

(b) T cells

(c) B cells

(d) Eosinophils

(e) Plasma cells

13. Macrophages may secrete p63 fig 2-12

(a) histamine (*mast cells, basophils, platelets*)

(b) serotonin (*platelets, mast cells in rodents*)

(c) **prostaglandins (all leukocytes)**

(d) **oxygen free radicals (?only in phagolysosome)**

(e) none of the above

14. Regarding chronic inflammation (2000)

(a) it characterised by hyperaemia, oedema, and leukocyte infiltration (*acute*)

(b) **monocytes use the same chemotactic pathways as neutrophils**

(c) it is *not always preceded by acute inflammation* (rheumatoid)

(d) may result in resolution (*not always*)

\***(e) angiogenesis is a feature of *wound healing and attempts at repair***

15. Regarding chronic inflammation, which is correct? (p79-82)

(a) ***Macrophages have a half-life of several months to years.***

(b) it is not always preceded by acute inflammation, *can be insidious in onset*

\***(c) unlike acute inflammation, *lymphocytes play a role in cell-mediated reactions,***  
and the production of immunoglobulin. They also stimulate macrophages, and  
macrophages stimulate them back, propagating chronic inflammation

\***(d) prolonged exposure to toxins such as silica causes *chronic inflammation***

\***(e) attempts at healing are evident**

16. Regarding chronic inflammation which of the following is FALSE? p79

(a) It is associated with persistent infections

(b) **It involves attempts at repair, rather than just tissue destruction**

(c) It may contribute to the formation of atherosclerosis

(d) It can be caused by exposure to toxic agents

(e) It involves mononuclear inflammatory cells