**Question 1**

Which of the chemical mediators of inflammation are produced form arachidonic acid and generates a febrile response?

A Histamine

B C5a

C Leukotriene

D Prostaglandin

Explanation D

This question can present as an EMQ

The lipid mediators prostaglandins and leukotrienes are produced from arachidonic acid (AA) present in membrane phospholipids and stimulate vascular and cellular reactions in acute inflammation.

In addition to their local effects PG are involved in the pathogenesis of PAIN AND FEVER in inflammation. PGE2 is hyperalgesic, and makes skin hypersensitive to painful stimuli such as intradermal injection of suboptimal concentrations of histamine and bradykinin. It is involved in cytokine induced fever during infections

Other stem options: C3B, serotonin, IL-6, NO, C5B, TNF

**Question 2**

All of the following infections cause granulomatous infections EXCEPT?

A Leprosy

B Lymphogranuloma venereum

C Schistosomiasis

D Syphilis

Explanation B

Causes of granulomatous disease

Infectious:

Bacterial: tuberculosis, leprosy, syphilis, cat-scratch disease

Parasitic: schistosomiasis

Fungal: Histoplasmosis; Blastomycosis

Inflammatory: temporal arteritis, chron’s disease, sarcoid,

Inorganic particulates: silicosis, berylliosis

Foreign body granuloma (suture or talc)

LGV is primarily an infection of lymphatics and lymphnodes. Chlamydia trachomatis is the bacterium responsible for LGV

Not a great question, don't know if the answer is correct. Form the current text book "The lesions of lymphogranuloma venereum contain a mixed granulomatous and neutrophilic inflammatory response." Left in the bank because it has appeared before.

**Question 3**

Which of the following is the first reaction in acute inflammation?

A Oedema

B Arteriolar dilation

C Arteriolar constriction

D Stasis of blood flow

Explanation C

Vasodilitation follows a transient constriction of arterioles that lasts a few seconds.

Note: the current textbook has changed the wording to "vasodilation is one of the earliest manifestations of acute inflammation; SOMETIMES it follows a transient constriction of arterioles lasting a few seconds". In the older editions, it did not say sometimes, but that vasoconstriction did occur a few seconds before vasodilatation.

Latest update: the current textbook: does not describe the transient arteriole constriction. It now describes the three components of acute inflammation- dilatation of small vessels, increased permeability and emigration of leukocytes.

Worth keeping the question, unlikely to be repeated

**Question 4**

Regarding chronic inflammation, which of the following statements is correct?

A Frequently resolves spontaneously

B Macrophages are the major cellular players

C It always follows acute inflammation

D Characterised by increased vascular permeability and oedema

Explanation B

Macrophages are the major cellular players and have a half life of several months to a year. Monocytes have a half life of one day. Acute inflammation is characterised by hyperaemia, oedema and leucocyte infiltration. Chronic inflammation is not always preceded by acute inflammation but may follow it. Chronic inflammation includes some of the most common and disabling human disease such as rheumatoid arthritis, tuberculosis and artherosclerosis

**Question 5**

Which of the following options is correct in relation to chronic inflammation?

A It is always preceded by acute inflammation

B It is characterised by hyperaemia, oedema and leucocyte infiltration

C Monocytes use the same chemotactic pathway as neutrophils

D The most frequent outcome is full resolution

Explanation C

Acute inflammation is characterised by hyperaemia, oedema and leucocyte infiltration. Chronic inflammation is not always preceded by acute inflammation but may follow it. Chronic inflammation includes some of the most common and disabling human disease such as rheumatoid arthritis, tuberculosis and artherosclerosis

Migration of monocytes is analogous to migration of neutrophils in acute inflammation

**Question 6**

In relation to compliment factor C5a, which of the following statements is false?

A Increases vascular permeability and causes vasodilation

B Is chemotactic for neutrophils

C Stimulates arachidonic acid (AA) metabolism

D Acts as an opsonin and favours phagocytosis by neutrophils

Explanation D

C5a, a powerful inflammatory mediator, is chemotactic for neutrophils, eosinpohiles and basophiles. C5a activates the lipoxygenase pathway of arachidonic acid (AA) metabolism in neutrophils and monocytes. C5a also plays a role in increased vascular permeability and chemotaxis. C3b and C3Bi, but not C5a, when fixed to bacterial cell walls act as opsonins and promote phagocytosis by neutrophils and macrophages.

**Question 7**

Regarding mast cells, which of the following statements is correct?

A They are only found in mucosal membranes

B They are derived from thymus

C They can degranulate without IgE

D ADP is a stimulator of mast cell degranulation

Explanation C

Mast cells are derived from the bone marrow and are widely distributed in the tissues. They are found predominantly near blood vessels, nerves and subepithelial sites. Mast cells participate in both acute and chronic inflammatory reactions. Mast cells release multiple primary and secondary mediators but not lysosomes. Adenosine triphosphate provides the energy for mast cell degranulation.

Other stimulators of mast cell degranulation are C5a, C3a, IL8 (interleukine 8 cytokine),drugs such as codeine, morphine, adenosine and mellitin along with physical stimuli

**Question 8**

Which of the following statements is correct in relation to bradykinin?

A Causes relaxation of smooth muscles

B Is formed from pre kallikrein

C Causes vasodilation

D Is not painful when injected into the skin

Explanation C

Kininogens form vasoactive peptides and the enzymes involved are kallikriens. Bradykinin causes increased vascular permeability, contraction of smooth muscle, dilatation of blood vessels and pain when injected into the skin

**Question 9**

Which of the following is released by macrophages?

A Serotonin

B Toxic oxygen metabolites

C IL-17

D Histamine

Explanation B

Factors released by macrophages include toxic oxygen metabolites, proteases, neutrophil chemotactic factors, coagulation factors, arachidonic acid (AA) metabolites (I would include PGs and Leukotrienes), nitric oxide, growth factors, angiogenesis and remodelling collagenases. PAF is also produced.

Others include: TNF, IL-1, IL-6, Chemokines, IL-12

**Question 10**

Which of the following is not chemotactic?

A Leukotriene B4

B Bradykinin

C Histamine

D Bacterial polypeptides

Explanation B

Other chemotactic factors include kallikrein, platlet activating factor (PAF), lysosomal proteins, chemokines and fibrinopeptides. Histamine is chemotactic for eosinophils.

Bradykinin direct effects are: smooth muscle contraction, arteriolar dilatation, increased permeability of venules, pain. Bradykinin is inactivated by kininases.

**Question 11**

Regarding phagocytosis, which of the following statements is correct?

A IgM is a potent opsonin

B Occurs in 2 steps: engulfment and killing

C C5a is an opsonin

D Bacterial killing occurs by mainly O2 dependant mechanisms

Explanation D

Phagocytosis occurs in three steps;

- recognition and attachment

- engulfment

- degradation

C5a is not an opsonin. IgG is a potent opsonin as is the C3b breakdown product and certain plasma lecitins. Phagocytosis is greatly enhanced by opsonisation

By itself, IgM is an ineffective opsonin; however it contributes greatly to opsonization by activating complement and causing C3b to bind to the antigen

**Question 12**

Regarding fatty change, which of the following statements is false?

A May result from diabetes mellitus

B May result from protein malnutrition

C Fatty acids are oxidised in the mitochondria

D May represent unmasking of normal cell fat content

Explanation D

Fatty change and steatosis describe abnormal accumulations of triglycerides within parenchymal cells. It occurs most often in the liver but also in the kidney, heart and muscles. Causes include alcohol (most common), diabetes mellitus (DM), protein malnutrition, toxins, obesity and anoxia. Fatty change per se, is reversible. Mild accumulation in an organ will not impair cellular function. Severe accumulation may impair function and even cause cell death.

**Question 13**

The first vascular response to injury is which of the following?

A Recruitment of vascular beds

B Arteriolar vasoconstriction

C Slowing of the circulation

D Capillary engorgement

Explanation B

Vasodilation follows a transient constriction of arterioles lasting a few seconds.

Note; The actual main change of the vessel in acute inflammation is vasodilation. However as you read the fine print, it SOMETIMES follows a transient constriction of the arterioles, lasting a few seconds.

Rather a tricky MCQ-but none of the stems say vasodilation-making it easier to answer

**Question 14**

Which statement is CORRECT regarding the movement of leucocytes towards the sire of injury?

A In response to C3b

B The process of transmigration is mediated by ICAM and PECAM integrins

C Predominantly as monocytes on the first day post injury

D Largely in the arterioles

Explanation B

Leukocyte migration through endothelium (transmigration or diapedesis) - occurs mainly in post-capillary venules. - Chemokines act on the adherent leukocytes and stimulate the cells to migrate through interendothelial spaces toward the chemical concentration gradient, that is, toward the site of injury or infection where the chemokines are being produced. - Several adhesion molecules present in the intercellular junctions between endothelial cells are involved in the migration of leukocytes. These molecules include a member of the immunoglobulin superfamily called PECAM-1 (platelet endothelial cell adhesion molecule), ICAM (intercellular adhesion molecule 1) or CD31and several junctional adhesion molecules. After traversing the endothelium, leukocytes pierce the basement membrane, probably by secreting collagenases, and enter the extravascular tissue. The cells then migrate toward the chemotactic gradient created by chemokines and accumulate in the extravascular site.

**Question 15**

Regarding chemical mediators of inflammation, which of the following options is correct?

A The kinin system is activated in platelets

B Histamine is derived from plasma

C C3b is within macrophages

D Serotonin is preformed in platelets

Explanation D

Histamine is derived from mast cells, basophils and platelets. C3b is derived from liver and formed in plasma. The kinin system is activated in plasma. Nitric oxide is produced in macrophages

**Question 16**

Chronic inflammation is characterised by which of the following options?

A Most frequently results in resolution

B Always preceded by acute inflammation

C Characterised by hyperemia, oedema and leukocyte infiltration

D The factors underlying monocyte infiltration are the same as for acute inflammation

Explanation D

Macrophages are the major cellular players and have a half life of several months to a year. Monocytes have a half life of one day. Acute inflammation is characterised by hyperaemia, oedema and leucocyte infiltration. Chronic inflammation is not always preceded by acute inflammation but may follow it. Chronic inflammation includes some of the most common and disabling human disease such as rheumatoid arthritis, tuberculosis and artherosclerosis.

**Question 17**

In the triple response, reactive hyperaemia is due to?

A Arteriolar dilitation

B Blushing

C Exercise

D Inflammatory mediators

Explanation A

The triple response consists of;

- Red line

- Wheal

- Flare

Hyperaemia and reactive hyperaemia is an active process due to arteriolar dilatation

**Question 18**

Macrophages are derived from which of the following?

A B-cells

B Monocytes

C T-cells

D Plasma cells

Explanation B

Macrophages are the dominant cellular player in chronic inflammation. Mononuclear phagocytes arise from a common precursor in the bone marrow, which give rise to the blood monocytes. From the blood, monocytes migrate into various tissues and differentiate into macrophages

**Question 19**

Which of the following cells are capable of lysing a cell without prior antigen sensitisation?

A Natural killer cells

B Neutrophils

C T-cells

D Macrophages

Explanation A

Natural Killer (NK) cells have the ability to kill a variety of both infected and tumour cells without prior exposure or activation by microbes and tumours. This ability makes the NK cells an early line of defence against viral infections and perhaps some tumours.

Extra: Neutrophils and macrophages are also part of the innate immune system, and therefore act independently of antigen presentation. Although not explicit in the text, neutrophiles and macrophages may be correct as well (just a thought!)

**Question 20**

A 55 year old man presents to the emergency department with a blood sugar of 26. His current medication is metformin. Which of the following is the correct hypersensitivity reaction?

A Type III hypersensitivity reaction

B Type IV hypersensitivity reaction

C Type II hypersensitivity reaction

D Type I hypersensitivity reaction

Explanation C

Type II = insulin resistant diabetes

Target antigen= insulin receptor

Mechanism of disease= antibody inhibits binding of insulin

Causes NIDDM-type 2 DM

Type IV= type I diabetes mellitus

Antigens of pancreatic islet B cells (insulin, glutamic acid decarboxylase, others)

Mechanism of disease insulitis (chronic inflammation in islets), destruction of B cells

Causes IDDM-type 1 DM