ACEM Primary Examination Vivas > Pathology > Inflammation
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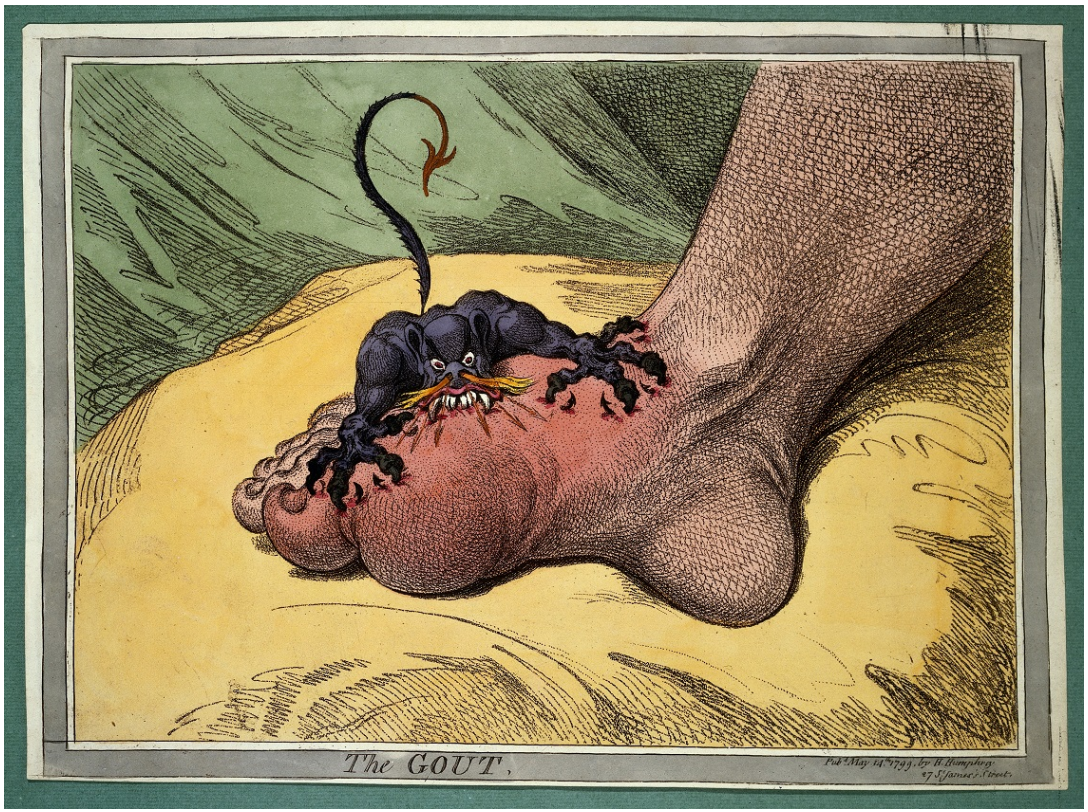

Acute Inflammation 2017-1-A

Stem: Moving onto Pathology. The most likely diagnosis is acute parotitis.			
Question 3 Inflammation **Subject**: Path LOA: 1	a) Describe the vascular changes in acute inflammation	a) • **Vasodilatation**: opening of arterioles and capillary beds mediated by histamine and nitric oxide (NO) leading to increased blood flow • **Increased vascular permeability** • **Stasis:** due to plasma protein permeability and increased viscosity	**all 3 bold** to pass
	b) What are the mechanisms responsible for increased vascular permeability in inflammation?	b) • Endothelial contraction / retraction: gaps in venules due to histamine, bradykinin and leukotrienes , causing immediate transient response (lasting 15 - 30 mins). Other stimuli (eg UV radiation, burns, some bact toxins) result delayed prolonged leakage (delay 2-12 hrs and may last hrs to days) • Direct vascular endothelial injury (eg. in severe burns, microbial toxin injury), rapid onset but may last days • Leukocyte mediated leakage: in venules and pulm capillaries, long lasting (hrs) • Trancytosis : increased transport of fluid and protein thru endothelial cells, VEGF increases number +/- size transport channels	2 out of 4 to pass
	c) Describe the role of complement in inflammation	c) >20 proteins (incl C1-9) – once activated, trigger cascade • Recruitment and activation of lymphocytes (C3a, C5a) – inflammation trigger • Formation Membrane Attack Complex (MAC) – causing cell lysis • Phagocytosis (C3b) – Phagocyte recognizes C3b bound to microbe.	2 out of 3 to pass

Acute Inflammation 2016-2-D

Question 4 Croup and Acute inflammation **Subject:** Path LOA: 1	a. What is croup? *Prompt: What is the effect on the airway* Prompt: *What viral agents cause croup*? b. Describe the main characteristics of acute inflammation. *Prompt: Describe the general characteristics of acute inflammation.*	1. **Acute laryngotracheobronchitis** in children: inflammatory/spasmodic **narrowing of the airway** produces barking cough, inspiratory stridor. Causes are predominantly viral, esp **Parainfluenza virus.** RSV, adenovirus and influenza are others. 2. Main characteristics of Acute Inflammation: A. Relatively rapid onset. B. Alterations in vascular calibre that **increase blood flow.** C. **Leaky microvasculature**: Structural changes in microvasculature that permit plasma proteins and leucocytes to leave circulation**.** This leads to oedema. D. **Emigration of leucocytes (esp neutrophils),** their accumulation at site of infection, and activation to eliminate offending agent. E. Duration of hours to days.	Bold Bold

Acute inflammation 2015-1-B

Stem: We will now move to Pathology. An MRI shows acute swelling of his cord in the region of his injury			
Question 2 Mediators of inflammation (pp 56-66) **Subject:** Path LOA: 1	What stimuli cause production of inflammatory mediators? What are the chemical mediators of acute inflammation and what are their actions?	Substances released from necrotic cells, microbial products, cell injury, mechanical irritation. **Histamine:** vasodilation, inc vasc perm, endoth activation **PG:** vasodilation, inc vasc perm **Leukotrienes:** inc vasc perm, chemotaxis, WC adhesion & activation **PAF:** vasodil, inc vasc perm, chemotaxis, WC adhesion, degran **Complement:** WC chemo and activation, vasodilat **Cytokines** (TNF, IL-1): endo activation (adhesion), fever, pain, hypotension, dec vasc resist **Chemokines:** chemotaxis, WC activation **Kinins:** inc vasc perm, vasodil, pain, sm m contraction	2 to pass. 4 to pass (including names and actions)

Acute Inflammation 2014-2-B

Stem: 85 year old man presents to your ED in urinary retention, the day after a prostate biopsy. On PR examination, his prostate is extremely tender and you suspect prostatitis. We will start with Pathology.

TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Question 1 Acute inflammation – questions to focus on acute inflammation not prostatitis specifically (as this is an LOA 3 topic) (pp 48-56)	1. What are the three major components of acute inflammation?	1. **Dilation of small vessels** leading to increase blood flow. 2. **Increased permeability of the microvasculature** enabling plasma protein and leucocytes to leave the circulation. 3. **Emigration of leucocytes from the microcirculation to the site of injury.**	Bold to pass Neutrophils predominate in the early inflammatory (6 – 24 hours) infiltrate and are later replaced by monocytes and macrophages (24 – 48 hours).
Subject: Path LOA: 1	2. How are leucocytes delivered to the site of injury? PROMPT: What are the three processes that leucocytes undergo to move from the blood to the site of injury?	This is a multistep process mediated and controlled by adhesion molecules and chemokines. 1) **Margination:** Occurs when leucocytes adopt peripheral position along the epithelium. Rolling (transient adherence mediated by selectins), activation and firm attachment (mediated by integrins) to the endothelium. 2) **Transmigration** (diapedesis): across the endothelium. Migration through interendothelial spaces typically in post capillary venules. 3) **Chemotaxis**: Leucocytes move toward the site of injury along a chemical gradient of chemoattractants, which can be exogenous or endogenous.	Bold to pass Polymerisation of actin at the leading edge of the cell establishes a "front wheel " drive in the direction of the injury
	3. Name some of the chemoattractants responsible for chemotaxis?	Most common exogenous agent **Bacterial products.** Endogenous: IL-8, **C5a**, and Leukotriene B4. All bind to specific receptors and promote polymerisation of actin.	Bold + 1
	4. What chemical mediators are responsible for pain, fever and tissue damage?	**IL-1,** TNF, Prostaglandins, Bradykinin, Neutrophil and Macrophage Lysosomal enzymes, Oxygen metabolites, NO.	Bold + 1

Acute inflammation 2012-2

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Morphologic patterns and outcomes of acute inflammation LOA: 1	1. What are the different types of acute inflammation? *Prompt: What are the morphological patterns of acute inflammation?*	**1.a. Serous inflammation:** thin fluid from plasma or mesothelial lining cells e.g. burns, effusions (pericardial, pleural) **b. Fibrinous inflammation:** more severe injuries and greater vascular permeability allows larger molecules such as fibrin e.g. characteristic of inflammation in body cavities (pericardial sac,meninges,pleura) **c. Suppurative / purulent inflammation:** large amounts of pus / purulent exudates – neutrophils,necrotic cells,oedema fluid e.g. organism type (staph) ; site (appendicitis) **d. Ulcers:** local defect in surface of an organ/tissue	2 with examples
	2. What are the outcomes of acute inflammation?	**2.a. Complete resolution +/- scarring** **b. Abscess formation** (suppurative inflammation) **c. Fibrosis** (fibrinous inflammation) **d. Chronic inflammation**	2 of 4

Acute inflammation 2008-2

1: Cellular changes in inflammation	Describe the sequence of cellular events in acute inflammation Prompts: • What cells are involved in acute inflammation? • How do these cells get from the blood vessels to the inflammatory site?	**Leucocytes are the major cell type involved. In first 6-24 hours neutrophils, and monocytes/macrophages in 24-48 hours** • Leucocytes line endothelial wall – margination **First stasis of blood flow leading to increased leucocytes along endothelial wall** **Then leucocyte adhesion to endothelial wall and diapedesis or transmigration across into interstitium – extravasation** • Adhesion and transmigration and recruitment are mediated by various mediators such as histamine, PAF cytokines and various attraction molecules – variously called immunoglobulins, integrins, selectins, mucin-like glycoproteins **Then leucocytes migrate to site of injury- chemotaxis** • Chemotaxis and activation is mediated thru various bacterial products, cytokines, chemical factors, Ag-Ab complexes products of necrosis **Then leucocyte activation to enable phagocytosis and enzyme release** Phagocytosis and release of various enzymes from leucocytes	Highlighted

Acute Inflammation, leucocytes 2011-2

Question 1 LOA: 1	1.What leukocytes types are characteristic of acute inflammation? (Prompt for 2)	**1. Neutrophils first 6-24 hours** Monocytes 24-48 hours Neutrophils may last longer (4 days) in pseudomonas Lymphocytes in viral Eosinophils in hypersensitivity	**Bold + 1 other**
	2. How do leucocytes get to an area of acute inflammation?	2. **Margination** of WCC in vessels, rolling and **Adhesion** to endothelium (pavementing) (Selectins) **Migration and diapedesis** across endothelium (PECAM1, CD31, Integrins) **Migration towards chemotactic** stimulus in tissue (bacterial products, cytokines, IL8, C5A)	**3 bold**
	3. Why do neutrophils predominate in the inflammatory response in the first 6-24 hours?	3. More numerous in the blood Respond more rapidly to chemokines May attach more firmly to adhesion molecules Neutrophils are short lived - disappear after 24-48 hrs (monocytes live longer)	**1/4**

Acute Inflammation, leucocytes 2010-2

Question 4.1 Cellular Events of Inflammation	1. How do leucocytes get to an area of acute inflammation? 2. What is the role of leukocytes in acute inflammation?	1.1 **Margination** of WCC in vessels, rolling and adhesion to endothelium (pavementing) (Selectins) 1.2 **Migration and diapedesis** across endothelium (PECAM1, CD31, Integrins) 1.3 **Migration towards chemotactic** stimulus in tissue (bacterial products, cytokines, IL8, C5A) 2 2.1 **Recognition and attachment** to materials (opsonins) mediated by receptors 2.2 Killing of microbes: **phagocytosis /engulfment /killing and degradation** (H2O2-MPO-Halide) 2.3 **Release of products** – Amplify the inflammatory reaction (lysosomal enzymes, reactive oxygen/nitrogen)	1. All Bold 2. 3/5 Bold

Acute inflammation, mediators 2012-2

Q2 Mediators of acute inflammation LOA: 1	1. What are the chemical mediators of acute inflammation? 2. What do they do? -	Histamine	Vasodilation, increased vasc permeability, endothelial activation	4 to pass 4 general correct actions
		Serotonin	Vasodilation, increased vasc permeability	
		Prostaglandins	Vasodilation, pain, fever	
		Leukotrienes	Increased vasc permeability, chemotaxis, leukocyte adhesion and activation	
		Platelet-activating factor	Vasodilation, increased vasc permeability, leukocyte adhesion, chemotaxis, degranulation, oxidative burst	
		Reactive oxygen species	Killing of microbes, tissue damage	
		Nitric oxide	Vascular smooth muscle relaxation, killing of microbes	
		Cytokines (TNF, IL-1)	Local endothelial activation (expression of adhesion molecules), fever/pain/anorexia/hypotension, decr vascular resistance (shock)	
		Chemokines	Chemotaxis, leukocyte activation	
		Complement (C5a, C3a, C4a)	Leukocyte chemotaxis and activation, vasodilation (mast cell stim	
		Kinins	Incr vasc permeability, smth muscle contraction, vasodilation, pain	
		Proteases activated during coagulation	Endothelial activation, leukocyte recruitment	

Acute inflammation, vascular changes 2013-1

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1 Vascular changes of acute inflammation	1. In acute inflammation what changes occur in blood vessels? Prompt: What happens next?	1. Changes in blood flow: (transient constriction), **vasodilation** (NO mediated) lead to **increased flow** • **Increased permeability,** loss of protein-rich fluid • Fluid loss & dilation lead to **stasis/congestion** • Leukocytes accum at vasc endothelium, endothelium expresses adhesion molecs, leuks adhere & migrate out	3/4 Bold
	2. What are the mechanisms for the increased vascular permeability seen in acute inflammation?	2. Chem mediated **endothelial cell contraction** (caused by eg histamine, LKT, sub P) • Endothelial injury direct/microbes/leuks eg burns • Increased transcytosis of fluids/proteins via channels of connected vesicles/vacuoles (vesiculovacuolar organelles) stim by factors eg VEGF	2/3 must include bold

Acute inflammation, vascular changes 2010-2

Question 1.1 Vascular Changes of Inflammation	1. Describe the vascular changes in acute inflammation 2. What are the mechanisms of increased vascular permeability?	1.1. **Vasodilatation:** opening of arterioles and capillary beds mediated by histamine and Nitric Oxide leading to increased blood flow 1.2. **Increased vascular permeability** 1.3. **Stasis:** due to PP permeability and increased viscosity 2. 2.1. Endothelial contraction / retraction: gaps in venules due to histamine and leukotrienes < 30mins, immediate transient response eg.ultraviolet radiation and kinins and leukotrienes 2-12hrs, delayed prolonged leakage eg. late appearing sunburn 2.2. Direct vascular endothelial injury eg. in severe burns, microbial toxin injury, amplified by neutrophil activation, rapid onset but may last days 2.3. Leukocyte mediated leakage, in venules and pulm capillaries, long lasting for hours 2.4. Trancytosis increased Tx of fluid and protein thru endothelial cell, VEGF	1. All 3 2. 2 out of 4

Acute inflammation, vascular changes 2009-1

Question 1: **Vascular changes of inflammation**	**Describe the vascular changes that occur in acute inflammation**	**1.Vasodilation & increased blood flow mediated by histamine and NO, action on vascular smooth muscle** **2. Increased permeability** 3.Stasis - incr blood viscosity and concentration of RBCs 4 Accumulation of leukocytes on vascular endothelium	Need 1 + 2 and 1 other
	What are the causes of the increased vascular permeability?	**1. Gaps due to endothelial contraction via mediators** ("immediate transient response"): histamine (fast), bradykinin, sub P, leukotrienes, cytokines(longer). Venules. 2. Direct injury to vessel: ("immediate sustained") 3. "Delayed prolonged" 2-12 hrs burn, radiation, toxins 4.. Leukocyte mediated injury: venules, pulm caps, hours 5. Incr transcytosis: vesicles, vacuoles, incr channels VEGF 6. New vessel formation; new bvs leaky; VEGF, mediators	Need 1 and 2 others

Acute inflammation, vascular changes 2007-1

2. Vascular changes of inflammation	Please describe the main vascular changes that occur with acute inflammation	**1.Vasodilation & incr blood flow via mediators eg histamine, NO action on vasc smooth muscle** **2. Increased permeability** 3.Stasis (mins) 4 Accumulation of leukocytes on vasc endothelium	Points 1 & 2 + 1 other
	What are the major mechanisms for the increased vascular permeability that occurs with inflammation? Prompt: "What mediators affect vascular permeability"	**1. Gaps due to endothelial contrac via mediators** (immediate transient): histamine (fast), bradykinin, sub P, leukotrienes, cytokines(longer). Venules. 2. Direct injury to vessel: immediate sustained eg burns, lytic bact 3. Delayed prolonged 2-12 hrs burn, radiation, toxins mech unclear 4.. Leukocyte-dep injury: venules, pul caps, hours 5. Incr transcytosis: vesicles, vacuoles, incr channels VEGF 6. New vessel formation; new bvs leaky; VEGF, mediators	Point 1 + 2 others

Acute inflammation, vascular changes 2006-1

TOPIC: Vascular changes of inflammation **NUMBER:** Question 1 – Friday 7 April am

OPENING QUESTION	**Describe the vascular changes in acute inflammation.**	**COMMENTS**
POINTS REQUIRED	1 Transient vasoconstriction sometimes.	2 to pass
	2 Vasodilatation, arterioles first then capillary beds resulting in increased local blood flow.	
	3 Increased vascular leakage \ permeability resulting in oedema formation and increased blood viscosity.	
	4 Circulatory slowing with stasis and neutrophil margination	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	**What mechanisms cause increased vascular leakage in acute inflammation?**	2 to pass
POINTS REQUIRED	1 Formation of gaps in venules due to endothelial contraction	
	2 Direct endothelial injury	
	3 Leukocyte-mediated injury	
	4 Increased transcytosis	
	5 Leakage from new blood vessels	
	6	
PROMPTS .		

COMMENTS

Standard opening question , should get most of question 1, then expect , two of the mechanisms in question 2 for a pass?

Acute inflammation, vascular changes 2003-2

2.1 Acute inflammation, vascular	Describe the vascular response in acute inflammation	Changes in vascular flow and caliber:-> transient constriction –> **dilation** –> heat and redness. **Increased vascular permeability**: -> slowing of circulation, hemoconcentration –> **stasis** –> leucocyte margination, adherence to endothelium -> oedema.	
	What are the mechanisms of increased vascular permeability in acute inflammation?	Vascular leakage – endothelial contraction; cytoskeletal reorganisation; direct injury; leucocyte injury; increased. transcytosis	3/

Chronic inflammation 2016-2-A

Stem: Moving to Pathology. His ulcer is chronic.			
Question 4 Chronic inflammation **Subject:** Path: LOA 1	1) What cell types are present in chronic inflammation?	**Macrophages** Multinucleate giant cells Lymphocytes Plasma cells Eosinophils Mast cells Neutrophil polymorphs (scarce)	*Bold PLUS 2 others*
	2) What processes mediate the persistent accumulation of macrophages seen in chronic inflammation?	1) **Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors- Macrophage Activation Factor) 2) Local proliferation of macrophages 3) Immobilisation of macrophages (Migration inhibition factor)	*Bold to pass*
	3) What clinical conditions can cause chronic inflammation? *Prompt: Anything other than--*	**Persistent infection:** Tuberculosis Syphilis Abscess Empyema Osteomyelitis **Prolonged exposure to an agent**: Exog –foreign body, persistent trauma, silica -> silicosis Endogenous – lipid -> atherosclerosis **Autoimmune:** Rheumatoid Arthritis Multiple Sclerosis Inflammatory Bowel Disease Systemic Lupus Erythematosus	*At least 3 examples from at least 2 categories*

Chronic inflammation 2013-2-B

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 PATHOLOGY LOA: 1	*"A patient presents with chronic inflammatory arthritis."* 1. What are the characteristics of chronic inflammation?	• Inflammation for a **prolonged period** (week or more). • Characterised by **macrophages**, lymphocytes and plasma cells • With simultaneous-active inflammation/ **tissue destruction** and **attempts at repair** by connective tissue, fibrosis	¾ Bold to pass
	2. Why does macrophage accumulation persist in chronic inflammation?	**Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors) Local proliferation of macrophages Immobilisation of macrophages	Bold
	3. What are the causes of chronic inflammation? (prompt can you give an eg. of each)	• **Persistent infection**- TB, syphilis • **Autoimmune**-RA, MS, IBD, SLE • **Prolonged exposure to an agent**: exogenous-silica->silicosis , FB, persistent trauma endogenous-lipid->atherosclerosis	2/3 bold with examples

Chronic inflammation 2011-2

Question 1 LOA: 1	1. What are the characteristics of chronic inflammation?	1.Inflammation for a **prolonged period** (week or more) Characterised by **macrophage** With simultaneous - active inflammation - **tissue destruction** - **attempts at repair**	**Bold**
	2. What are the causes of chronic inflammation? *Prompt: Can you give an example of each of these?*	2.**Persistent infection** TB, syphilis, PUD **Prolonged exposure toxic agents** exogenous = silica / FB endogenous = lipid - atherosclerosis **Autoimmune** RA; lupus	**2/3 Bold with one example**
	3. Why does macrophage accumulation persist in chronic inflammation?	**Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors) Local proliferation of macrophages Immobilisation of macrophages	**Bold**

Chronic inflammation 2009-2

Qn 2 ACEM PRIMARY 2009/2 ANSWERS

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2: Chronic inflammation	(a) What cell types are present in chronic inflammation?	**Macrophages** Lymphocytes Plasma cells Eosinophils Mast cells Neutrophils	Bold plus 2 others to pass
	(b) What processes mediate the persistent accumulation of macrophages seen in chronic inflammation?	1. **Continued recruitment** of monocytes (continued expression of adhesion molecules and chemotactic factors) 2. Local proliferation of macrophages 3. Immobilisation of macrophages	Bold to pass
	(c) What products are released by activated macrophages in chronic inflammation?	**Products associated with tissue injury:** • Toxic O_2 metabolites; Proteases (elastases, collagenases); Neutrophil chemotactic factors; Coagulation factors; AA metabolites; Nitric oxide **Products associated with fibrosis:** • Growth factors (PDGF, FGF, TGF); Fibrogenic cytokines; Angiogenesis factors (FGF); "Remodelling" collagenases	Processes in bold and an example of each Simple list (of 5 or more) passes. Better pass if organised into groups

Chronic inflammation 2007-2

TOPIC: Chronic Inflammation _____ **NUMBER: Q1** _____

OPENING QUESTION	1) What are the defining pathological characteristics of chronic inflammation ?	**COMMENTS**
POINTS REQUIRED	a) **infiltration with mononuclear cells** - including macrophages, lymphocytes and plasma cells as a reaction to persistent injury b) **tissue destruction** largely induced by the inflammatory cells c) attempts at **repair by connective tissue replacement** (angiogenesis and fibrosis) Prompt: What are the morphological features of chronic inflammation	Pass / Fail must mention 3 phases
SECOND QUESTION	List some causes of chronic inflammation	
POINTS REQUIRED	• Persistent infection Syphilis, TB • Toxic agents eg silica or beryllium • Autoimmunity eg rheumatoid, lupus	2 of 3 components

Complement 2008-2

1. Role of complement in inflammation	1. What is the complement system?	**Plasma protein system involved in immunity** against microbes. Complement proteins numbered C1-9 are present in plasma in inactive forms.	Highlighted
	2. Describe the main pathways by which complement activation occurs.	1. **Classical pathway:** involving an antigen-antibody complex 2. **Alternate pathway:** triggered by microbial surface molecules (e.g. endotoxin). No antibody involvement. 3. Lectin pathway: plasma mannose-binding lectin binds to carbohydrate on microbe All pathways result in cleavage and activation of C3 (most important and abundant complement component)	Highlighted & way activated
	3. How do activated complement products mediate acute inflammation?	1. **Vascular effects**: increased permeability; vasodilatation (via C3a, C5a mediated histamine release from mast cells) 2. Leucocyte adhesion, chemotaxis and activation: via C5a 3. Phagocytosis: C3b acts as opsonin on microbe and leads to phagocytosis 4. Cell lysis by the membrane attack complex (MAC) – composed of multiple C9 molecules	Vascular and one other

Complement 2006-2

TOPIC: Role of complement in inflammation _____ **NUMBER:** _____

OPENING QUESTION	Describe the role of complement in inflammation.	**COMMENTS**
POINTS REQUIRED	1 Vascular phenomena	3/3 to pass
	2 Leucocyte adhesion, chemotaxis and activation	
	3 Phagocytosis	
PROMPTS		
SECOND QUESTION (if needed)	Of the complement components, which are the most important inflammatory mediators	Must name both
POINTS REQUIRED	1 C3	
	2 C5	
PROMPTS		

Complement 2003-1

TOPIC: Complement in Immune Processes **NUMBER:** _____

OPENING QUESTION	What is the role of complement in systemic immune complex disease	**COMMENTS**
POINTS REQUIRED	1 Opsonisation (c3b)	3 of 4, not actual numbers to pass
	2 Chemotaxis (c5 & b67)	
	3 Anaphylotoxins (c3a & 5a)	
	4 Membrane Attack Complexes (c5-9)	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	What is an Arthus Reaction	
POINTS REQUIRED	1 Localised	
	2 Excess of antibody	
	3 Large immune complexes – precipitates - vasculitis	
	4	
	5	
	6	
PROMPTS		

Mediators of Inflammation

Question 2.
 	
Cell derived mediators of inflammation
	1. Which mediators of inflammation are derived from cells?	• Preformed
		  ○ **Vasoactive amines**
		  ▪ **Histamines**
		  ▪ Serotonin
		• Newly synthesized
		  ○ **Arachidonic metabolites**
		  ▪ Prostaglandins
		  ▪ Leukotrienes
		  ▪ Lipoxins
		  ○ Reactive Oxygen Species
		  ○ Platelet activating factors
		  ○ Nitric Oxide
		  ○ Cytokines (TNF, IL1)& Chemokines
			Pass = bold + 1 other

	2. Which cells release histamine?	Widely distributed in tissues, richest sources: • **Mast cells** • **Basophils** • **Platelets**	**Pass =/> 2**
	3. What **are the effects of histamines in an inflammatory response?**	• **Dilation of the arterioles** • **Increased vascular permeability of the venules** • Can cause constriction of large arteries	**Pass = bold (2)**