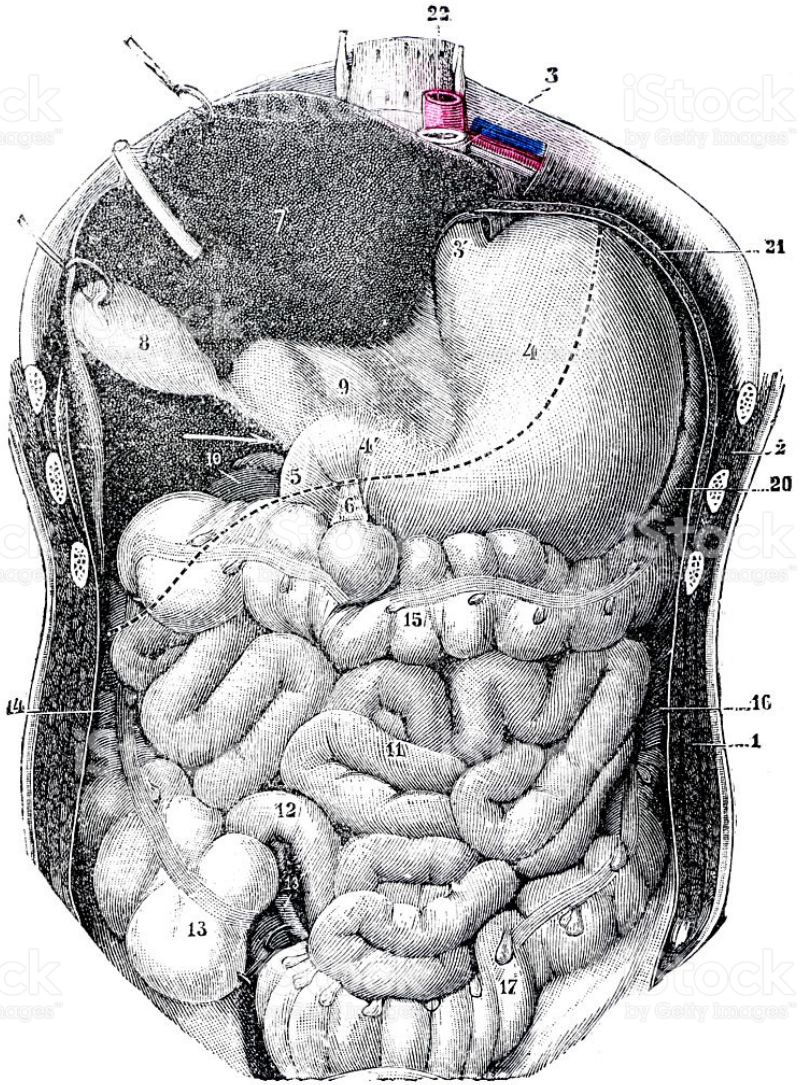
| **ACEM Primary Examination Vivas > Pathology > GI**<br>Organised by edvivas.com | |
|---|---|
| **Title** | **Page** |

Bowel Obstruction 2016-2-D

| | | | |
|---|---|---|---|
| **Stem:** Moving onto Pathology. | | | |
| **Question 4**<br>Intestinal obstruction<br><br>**Subject:** Pathology<br>LOA: 2 | a. Describe the common causes of bowel obstruction | Adhesions, hernia, malignancy, volvulus, intussusception, mesenteric infarct, strictures (due to Crohns, radiation, mesenteric ischaemia) | 4 of 7 |
| | b. How does a hernia form, and cause a bowel obstruction? | **Weakness/defect in abdominal wall**, protrusion of serosa lined pouch of peritoneum (hernia sac).<br>**Visceral protrusion** (small bowel, large bowel, omentum most often involved.)<br>**Entrapment of hernia sac in a narrow neck** causes pain.<br><br>Ongoing obstruction → venous stasis, oedema → incarceration and strangulation<br>Common locations (inguinal, femoral, scars, umbilical) | Bold + 2 others |
| | c. Describe some important clinical sequelae of ongoing bowel obstruction | **Intestinal perforation**, **intestinal ischaemia** peritonitis, sepsis, abscess, electrolyte disturbance, vomiting and aspiration, death | Bold |

Cholera 2010-2

| | | | |
|---|---|---|---|
| Question 2.4<br><br>Cholera | 1. What is the causative organism of cholera?<br>2. Describe the pathogenesis of cholera (Describe how the enterotoxin causes diarrhoea). | 1. **Vibrio cholera = gram neg bacteria** (comma shaped/flagellate)<br><br>2. Pathogenesis<br>2.1. **Non invasive**<br>2.2. **Flagella proteins for attachment & colonization**<br>2.3. **Preformed enterotoxin**<br>2.3.1. Cholera enterotoxin<br>• 5 B subunits<br>• 1 A subunit<br>2.3.2. B subunit **binds to intestinal (mainly duodenum/jejunum) – epithelial cells**<br>• Retrograde transport in ER<br>2.3.3. A subunit Tx to cytoplasm<br>• A subunit activates G protein<br>• Stimulates adenyl cyclase→ c-amp<br>• Opens cystic fibrosis transmembrane conductance regulator (CFTR)<br>• **Releases $Cl^-$ into lumen**<br>○ **secretion of HCO3, Na and $H_20$**<br>○ **massive diarrhoea which overwhelms colonic resorption** | 1. Bold<br><br>2. Need 4 bold to pass |

Cholera 2006-1

**TOPIC:** Thursday PM – Q5 - Cholera _____ **NUMBER:** _____

| | | |
|---|---|---|
| **OPENING Qn** | What is the causative organism of cholera? | **COMMENTS** |
| **POINTS Req'd** | 1. Vibrio cholerae | |
| | 1. Gram –ve, flagellate , water-borne comma-shaped bacterium | |
| | 2. | |
| **SECOND Qn** | How does the organism cause diarrhoea? | |
| **POINTS Req'd** | 1.Non-invasive in gut lumen | |
| | **2. Release of enterotoxin** | |
| | **3. Secretory diarrhoea** | |
| | 4. Acts on G proteins | |
| | | |

Cholera 2004-2

**TOPIC:** Cholera **NUMBER: 3**

| | | | |
|---|---|---|---|
| **OPENING QUESTION** | What causes cholera? | **PROMPTS** | **COMMENTS** |
| **POINTS REQUIRED** | 1. Cholera is caused by Vibrio cholerae which is a gram-negative bacteria<br>2. Vibrio cholerae produces enterotoxins known as Cholera toxins | Organism | |
| | 3. There is no epithelial cell invasion. | | |
| **SECOND QUESTION** | How does Vibrio Cholerae cause diarrhoea? | | |
| **POINTS REQUIRED** | Cholera toxin binds with the G-protein in the epithelial cells which stimulates adenyl cyclase and increase intracellular cyclic AMP, resulting in the massive secretion of chloride, sodium and water. The massive watery diarrhoea overwhelms the reabsorptive function of the colon. This results in litres of dilute "ricewater" diarrhoea with few leukocytes produced. There is massive sodium, chloride and bicarbonate and fluid losses. | Need to say it's due to Toxin, cause rice water diarrhoea | |

Chronic Gastritis 2011-1

| | | | | |
|---|---|---|---|---|
| **Question 4.**<br><br>**Chronic gastritis** | 1. What are the **causes of chronic gastritis?** | • **H Pylori**<br>• Chronic bile reflux<br>• NSAIDS<br>• Autoimmune<br>• Allergic response<br>• Infections<br>• Radiation | • Mechanical<br>• Psychological stress<br>• Chronic irritants (coffee, alcohol, caffeine )<br>• Systemic disease<br>• (Crohns, amyloid, graft vs host) | **H pylori** + 2 others |
| | 2. Describe the features of **H pylori induced chronic gastritis** | • Most **common cause**<br>• predominantly **antral**<br>• **High acid** production<br>• **Hypogastrinaemia**<br>• Generates **ammonia** (specific test)<br>• **Disruption normal mucosal defence** mechanisms | | 2/5 |
| | 3. What are the **complications of gastric ulcer?** | • Bleeding (15-20%)<br>  ○ Accounts for 25% of ulcer deaths<br>• Perforation<br>• Obstruction<br>• Gastric adenocarcinoma (complication of chronic H. Pylori pangastritis) | | 2/3 |

Chron's Disease 2009-1

| | | | |
|---|---|---|---|
| Question 5:<br>**Crohn disease** | **What are the pathological features of Crohn disease?** | **1. Transmural inflammation of bowel with skip lesions**<br>**2. Noncaseating granulomata**<br>**3. Fissures and fistulae** | 2/3 Bold needed |
| | **What are the extraintestinal manifestations of Crohn disease?** | Migrating polyarthritis, sacroiliitis, ank spondylitis, erythema nodosa, finger clubbing, sclerosing cholangitis (uncommon), Uveitis, mild hepatic pericholangitis, renal disorders due to trapping of the ureters (uncommon). Systemic amyloidosis (rare) GI tract cancer (less common than UC). May occur prior to intestinal symptoms. | At least three systems<br><br>Prompt:<br>What other inflammatory conditions may be seen in Crohn disease? |

Chron's Disease 2005-2

**TOPIC:** Crohn's Disease **NUMBER: 4a**

| | | |
|---|---|---|
| **OPENING QUESTION** | What are the characteristic pathological features of Crohn's Disease? | **COMMENTS** |
| **POINTS REQUIRED** | 1 Mouth-anus | 4 of 6 for pass |
| | 2 Skip lesions | |
| | 3 Chronic, relapsing | |
| | 4 Non-caseating granulomata | |
| | 5 Mucosal ulceration | |
| | 6 Transmural inflammation | |
| | * Comparison of Crohns and UC acceptable | |
| **PROMPTS** | | |
| **SECOND QUESTION** (if needed) | What are the complications of Crohn's Disease? | |
| **POINTS REQUIRED** | 1 Strictures | 2 of first 3 and 1 extra-intestinal manifestation for pass |
| | 2 Fistulae | |
| | 3 Malabsorption syndromes | |
| | 4 Extra-intestinal manifestations-polyarthritis, ank. Spond.; erythema nodosum, clubbing; pericholangitis, etc | |
| | 5 Neoplasia, less common than UC | |
| | 6 | |
| **PROMPTS** | What are the extra-intestinal manifestations of Crohn's Disease? | |

E. coli 2017-2-B

| **Stem:** A 90 year-old man with a urinary catheter presents with sepsis. Previous cultures have grown E.coli. We will start with Pathology. | | | |
|---|---|---|---|
| TOPIC | QUESTIONS | KNOWLEDGE **(essential in bold)** | NOTES |
| **Question 1**<br><br>E.coli<br><br>**Subject:** Pathology<br><br>LOA: 1<br><br>*Robins 9th Ed; Pg 350; Pg 665; Chapter 14:* | a) Which bacterial class does Escherichia coli belong to? | a) E.coli is a **gram negative rod** which is a facultative **anaerobe**. It is a normal GI pathogen. | Bold |
| | b) What is the difference between an endotoxin and an exotoxin? | b) Endotoxins are lipopolysaccharides (LPS) in the outer membrane of the **cell wall of Gram-negative bacteria which cause injury via the host immune response**<br><br>Exotoxins are proteins that are **secreted by the bacterium and cause direct injury** | Bold concepts |
| | c) List some types of infections that can be **commonly** caused by E.coli? | c) Urinary tract infections; prostatitis; epididymo-orchitis; infectious enterocolitis; cholecystitis; bacterial peritonitis | 3 to pass from this list |

E. coli 2010-2

| | | | |
|---|---|---|---|
| Question 4.5<br><br>E. coli Gastroenteritis | List the types of E. Coli enteritis and describe their features | **1.1 Enterotoxic E coli** (ETEC)<br>1.1.1 Food and water, traveller's<br>1.1.2 **LT heat labile toxin**, adenyl cyclise -> **inc cAMP -> inc Cl- secretion and decr absorption (cholera like)**<br>1.1.3 ST **heat stable toxin**, guanylate cyclase -> **incr cGMP**<br>**1.2 Enterohaemorrhagic E coli** (EHEC)<br>1.2.1 **Beef esp. ground**, milk vegetable<br>1.2.2 **O157:H7** and non O157:H7<br>1.2.3 **Shigella like toxin**<br>1.2.4 Large outbreaks, **bloody diarrhoea, haemolytic uraemic syndrome**<br>1.2.5 Thrombotic Thrombocytopenic purpure (2%)<br>**1.3 Enteroinvasive E. Coli** (EIEC)<br>1.3.1 Food, water, person to person<br>1.3.2 No toxins, invades mucosa, colitis<br>**1.4 Enteroaggregative E. coli** (EAEC)<br>1.4.1 Adheres via **adherence fimbriae.**<br>1.4.2 Dispersin (removes –ve charge/ protection)<br>1.4.3 Shigella like toxin and ETEC ST toxin<br>1.4.4 Non bloody diarrhoea, prolonged in AIDS | 2 of 4 groups to pass<br><br>1 feature of any two |

Gastroeneritis 2004-2

**TOPIC:** Gastroenteritis **NUMBER: 3**

| | | | |
|---|---|---|---|
| **OPENING QUESTION** | What are the common causes of infective gastroenteritis? | **PROMPTS** | **COMMENTS** |
| **POINTS REQUIRED** | 1. Viral: Rotavirus (group A), Norwalk like virus, Enteric adenovirus | Prompt to give examples, need viral & bacterial causes to pass | |
| | 2. Bacterial:<br>❖ Ingestion of preformed toxin: Stahpylococcus aureus, Vibrios & Clostridium perfringens.<br>❖ Toxigenic organism: proliferate within the lumen & form enterotoxin: E coli, Vibrio cholerae.<br>❖ Enteroinvasive organism: proliferate, invade & destroy mucosal epithelial cells: E coli, Shigella, Salmonella. | | |
| | 3. Parasites: Giardia Lamblia, Entamoeba histolytica. | | |

Infective Enterocolitis 2013-1

| | | | |
|---|---|---|---|
| Question 4<br>Infective enterocolitis<br>LOA: 2 | 1. What are the organisms that cause infectious enterocolitis? | **1. Bacterial**- E.coli, Salmonella, Shigella, Campylobacter, C.difficile, Cholera, Yersinia, Mycobacteria<br>**Viral**- Norovirus, Rotavirus, Adenovirus<br>Parasitic- Giardia, Amoeba, Cryptosporidium, other (nematodes, cestodes, trematodes) | Bold with 1 bact & 1 viral<br>3 examples total |
| | 2. What is pseudomembranous colitis? | 2. Colitis caused by **overgrowth of C. difficile** ( also Salmonella, C.perfringens typeA, S.aureus)<br>Associated with antibiotic use<br>Forms a **pseudomembrane** made up of adherent layer of inflammatory cells and debris | Bold |
| | 3. What are the risk factors for development of pseudomembranous colitis? | 3. **Risk factors- advanced age, hospitalisation, antibiotic treatment** | 2/3 Bold |
| | What are the clinical features of pseudomembranous colitis? | 30% hospitalised patients colonised, but most aymptomatic<br>**Fever**, leucocytosis, **abdominal pain**, cramps, hypoalbuminaemia, watery **diarrhoea**, dehydration, rarely gross bloody diarrhoea<br>Diagnosis-usually detection of toxin<br>Treat with metronidazole, vancomycin | Bold |

Ischaemic Bowel Disease 2014-2-C

| **Stem: We will now move on to Pathology.** On examination she has a rigid, tender abdomen. | | | |
|---|---|---|---|
| **Question 4**<br>Ischaemic Bowel<br>(pp 791-793)<br><br>**Subject:** Path<br><br>LOA: 1 | What conditions can lead to infarction of bowel | **1. Acute arterial obstruction**<br>Atherosclerosis, Aortic aneurysm, Hypercoagulable state, OCP use, Embolism<br>**2. Intestinal hypoperfusion**<br>cardiac failure, shock, dehydration, vasoactive drugs<br><br><u>systemic vasculitis</u><br>HSP, Wegeners granulomatosis<br><br><u>Mesenteric venous thrombosis</u><br>Hypercoagulable state, Invasive neoplasm, Cirrhosis, Trauma, Abdominal masses<br><br><u>Miscellaneous</u><br>Radiation, Volvulus, Stricture, Amyloid, diabetes | **BOLD to pass**<br>**Minimum 2 from each bolded group**<br><br>**2 from non-bolded** |
| | What are the clinical features of ischaemic bowel? | Severe **pain**, may be transient. Tenderness, peritonism, nausea, vomiting, bloody diarrhoea, melaena, shock, hyper/hypothermia, sepsis | **Bold + 3 features** |
| | What parts of the bowel are most susceptible to ischemic injury<br>And why? | **Watershed zones**<br>- Splenic flexure, sigmoid colon, rectum<br>- Located at end of arterial supply<br>Surface epithelium : Villi more at risk than crypts<br>Intestinal capillaries run from crypts up villi to surface | **Must be able to explain why watershed zones are most at risk** |

Ischaemic Bowel Disease 2011-1

| | | | | |
|---|---|---|---|---|
| **Question 4.**<br><br>**Ischaemic bowel disease** | **1. What conditions can lead to infarction of bowel?**<br><br>**PROMPT; by what mechanisms do these conditions cause injury** | **Acute vascular obstruction**<br>-atherosclerosis (esp. origin major vessels)<br>-aortic aneurysm<br>-hypercoagulable states<br>-OC use<br>-embolism<br>**Intestinal hypoperfusion**<br>-cardiac failure<br>-shock<br>-dehydration<br>-vasoconstrictive drugs | Systemic vasculitis<br>-Henoch-Scholein purpura<br>-Wegener's granulomatosis<br>Mesenteric venous thrombosis<br>-hypercoagulable states<br>-invasive neoplasms<br>-cirrhosis<br>-trauma<br>-abdominal masses | Bolded headings with 4 clinical examples to pass |
| | 2. **Describe the intestinal response to an acute ischaemic insult.**<br>Prompt: what is the mechanism by which ischaemic bowel injury occurs? | • Initial hypoxic injury<br>• **Secondary reperfusion injury**<br>- major injury in this phase<br>- free radical production, neutrophil infiltration, inflammatory mediator release<br>• Magnitude of response determined by<br>- vessels affected<br>- timeframe over which ischaemia develops | | Must know that it is predominantly a **reperfusion type injury** |
| | 3. **Which parts of the bowel are most susceptible** to acute ischaemic injury and **why**? | **Watershed zones**<br>-splenic flexure, sigmoid colon and rectum<br>-located at end of arterial supply<br><br>Surface epithelium: Villi more at risk than crypts<br>-intestinal caps run from crypts up villi to surface | | Must be able to explain why **watershed zones are most susceptible** to injury. |

Ischaemic Bowel Disease 2009-2

| TOPIC | QUESTION | ESSENTIAL KNOWLEDGE | NOTES |
|---|---|---|---|
| Question 4:<br><br>Ischemic bowel | 1. What are the predisposing conditions for the development of ischemic bowel?<br><br>Non-occlusive ischaemia<br>• cardiac failure<br>• shock<br>• dehydration<br>• vaso constrictive drugs<br>Miscellaneous<br>• radiation<br>• volvulus<br>• stricture<br>• amyloid<br>• diabetes<br>• internal or external herniation | **Arterial thrombosis**<br>• artherosclerosis.<br>• vasculitis<br>• aortic dissection<br>• iatrogenic – angiography or aortic reconstruction<br>• Hypercoagulable state.<br>• Oral Contraceptive Pill<br>**Arterial embolism.**<br>• SBE<br>• Angiography<br>• Aortic atheroembolism<br>**Venous Thombosis**<br>• Hypercoagulation<br>• OCP<br>• AT III deficiency.<br>• Intraperitoneal sepsis<br>• Post-operative<br>• Invasive neoplasms<br>• cirrhosis<br>• abdominal trauma | Simple list of 6 or more must contain examples of each of first 3 categories = straight pass<br><br>headings + good examples of each = better pass. |
| | 2. What are the clinical features of transmural infarction? | Pain<br>Tenderness<br>Nausea<br>Vomiting<br>Bloody diarrhoea, melanotic stool<br>Shock<br>Vascular collapse<br>Absent bowel sounds<br>Abdominal rigidity | Pain + any other 3 to pass |

Ischaemic Bowel Disease 2005-2

**TOPIC:** Ischaemic bowel _____ **NUMBER:** ~~4b~~ 4c .

| | | |
|---|---|---|
| **OPENING QUESTION** | What are the factors that predispose to ischaemic bowel disease | **COMMENTS** |
| **POINTS REQUIRED** | 1 Arterial thrombosis | 3of 5 for pass, must include arterial embolism |
| | 2 Arterial embolism, association with AF | |
| | 3 Venous thrombosis | |
| | 4 Non-occlusive ischaemia eg shock, drugs | |
| | 5 Miscellaneous eg radiation, herniae, vasculitis | |
| **PROMPTS** | | |
| **SECOND QUESTION** (if needed) | Regarding acute transmural infarction caused by arterial occlusion, describe the pathological course | |
| **POINTS REQUIRED** | 1 Congestion | 4 of 7, but must have perforation and metabolic |
| | 2 Oedema and haemorrhage in wall | |
| | 3 Lumen contains bloody fluid | |
| | 4 Mucosal necrosis | |
| | 5 Gangrene | |
| | 6 Metabolic and cardiovascular derangements eg acidosis/ fluid balance | |
| | 7 Perforation ..often leading to death | |
| **THIRD QUESTION** (if needed) | What are the outcomes of chronic bowel ischaemia? | Optional question |
| | Strictures –submucosal involvement | 1 of 2 for pass |
| | Segmental, patchy mucosal degeneration | |

Peptic Ulcer Disease 2008-1

| | | | |
|---|---|---|---|
| Q4. Peptic ulcer disease. | | | |
| | By what mechanisms may Helicobacter pylori cause peptic ulcers? | *1.H. pylori* secretes **urease**, which generates free ammonia; and a **protease** which breaks down glycoproteins in the gastric mucosa.<br>2. *H.* pylori makes **phospholipases** →damage surface epithelial cells glycoprotein complexes.<br>*3.H.* pylori enhances gastric secretion and impairs duodenal bicarbonate secretion. This enhances metaplasia.<br>4. Several H. pylori proteins are immunogenic→evokes **strong immune response** in the mucosa. Activated T and B cells are both seen in chronic gastritis caused by *H. pylori.*<br>5. Thrombotic occlusion of surface capillaries is promoted by a **bacterial platelet activating factor.**<br>6. Other antigens (including **lipopolysaccharide**) recruit inflammatory cells to the mucosa.<br>7. Damage to the mucosa is thought to permit **leakage of tissue nutrients** into the surface microenvironment, thereby sustaining the bacillus. | **Prompt:** What does *H. pylori* produce which can help cause ulceration?<br>Pass criteria: need to say it involves immunogenic response. |
| | What complications may arise from peptic ulcer disease? | 1. **Bleeding** (15-20%of patients), →25% of ulcer deaths<br>2. **Perforation**<br>~ 5% of patients<br>→2/3 of ulcer deaths<br>3. **Obstruction** from oedema and or scarring<br>~ 2% of patients<br>Mostly due to pyloric channel ulcers<br>Rarely causes complete obstruction with intractable vomiting &<br>incapacitating, crampy abdominal pain | Pass criteria: 2/3 |

Psuedomembranous Colitis 2010-2

| | | | |
|---|---|---|---|
| Question 3.3<br><br>Pseudo-membranous Colitis | 1. Describe the pathogenesis of pseudomembranous colitis.<br><br>2. What are the clinical features of pseudomembranous colitis?<br><br>3. What is the pseudomembrane? | **1.1. Disruption of normal bowel flora (ab's – esp. 3rd gen ceph) allowing overgrowth of *C. difficile***<br>**1.2. *C. difficile* elaborates toxins that cause:**<br>1.2.1. Ribosylation of small GTPases<br>1.2.2. Disruption of epithelial cytoskeleton<br>1.2.3. Tight junction barrier loss<br>1.2.4. Cytokine release<br>1.2.5. Apoptosis<br>1.3. Denuded surface epithelium<br>1.4. Superficial lamina propria contains dense infiltrate of neutrophils & occasional fibrin thrombi in capillaries<br>1.5. Damaged crypts are distended by mucopurulent exudates that erupt "volcanically"<br>1.6. Coalesce to form the pseudomembrane<br>2. Causes fever, (leukocytosis), profuse watery diarrhoea, abdo pain<br>**3. Pseudomembrane is an adherent layer of inflammatory cells and debris at sites of colonic mucosal injury** | 1. Toxin + one otherbold + 1 other (1.3 to 1.7)<br><br>2. 2/3<br>3. bold |

Salmonella 2011-2

| | | | |
|---|---|---|---|
| Question 3<br><br>LOA: 1 | 1. What type of bacterium is Salmonella? | 1. **Gram-ve bacillus** (Enterobacteriaceae family) | 1. Bold |
| | 2. Describe the pathogenesis of typhoid fever? | 2. Caused by **Salmonella typhi** (endemic) and **paratyphi** (travellers).<br>Endemic in India, Mexico, Phillipines, Pakistan, El Salvador, Haiti.<br>Taken up by mononuclear cells in the underlying lymphoid tissue in **gut invades** M cells<br>Reactive hyperplasia in lymph tissue.<br>**Disseminates by blood** | 2. Bold |
| | 3. What are the clinical features | 3. Causes fever, anorexia, vomiting and bloody diarrhoea.<br>BC +ve in 90% with fevers<br>Subsequent bacteraemia with fever and flu-like symptoms | 3. Reasonable response with prompting |
| Question 4 | 1 What is the pathogenesis of Type 2 | 1 **Insulin resistance** | Bold to pass |

Salmonella 2006-1

**TOPIC:** Friday AM – Q 5 – Salmonella dysentry _____ **NUMBER:** _____

| | | |
|---|---|---|
| **OPENING QUESTION** | What is the causative organism of salmonella dysentery? | **COMMENTS** |
| **POINTS REQUIRED** | 1. Salmonella enteritidis, typhimurium | |
| | 2. Gram negative, flagellated | |
| | | |
| **PROMPTS** | | |
| **SECOND QUESTION** (if needed) | What is the pathogenesis of salmonella dysentery? | |
| **POINTS REQUIRED** | 1. Invades epithelium (low oxygen environment) | 1 plus inflammation |
| | 2. Taken up by macrophages | |
| | 3. Gut wall inflammation | |
| | 4. Neural reflex pathway | |

Salmonella 2004-2

**TOPIC:** Salmonella Enteritis _____ **NUMBER: 3**

| | | | |
|---|---|---|---|
| **OPENING QUESTION** | Describe the Salmonella bacterium. | **PROMPTS** | **COMMENTS** |
| **POINTS REQUIRED** | Salmonella are flagellated, gram-negative bacteria. S enteritidis, S typhimurium and others cause food borne and water borne gastroenteritis. | Need to describe the type of bacteria | |
| **SECOND QUESTION** | How does Salmonella cause gastroenteritis? | | |
| | Salmonella pass through intestinal epithelial cells via transcytosis with minimal epithelia damage in the terminal ileum and colon | Need to say it's an invasive infection and say location | |
| | Salmonella enters into lamina propria that leads to 5-10% bacteraemia. | | |

Ulcerative Colitis 2010-2

| | | | |
|---|---|---|---|
| Question 2.3<br><br>Ulcerative Colitis | 1. What are the pathological features of Ulcerative Colitis? | **1.1.** One of two disorders that compromise **inflammatory bowel disease (IBD)**<br>**1.2.** Severe ulcerating inflammatory disease<br>**1.3.** Limited to **colon and rectum.**<br>1.3.1. Continuous distribution (Starts in colon and extends continuously – No skip lesions)<br>1.3.2. Extends only into mucosa and submucosa (ie not trans mural)<br>1.3.3. Pancolitis if entire colon affected, limited distal disease eg ulcerative proctitis<br>1.4. Superficial broad based ulcers<br>1.5. Pseudopolyps<br>1.6. Malignant potential<br>1.7. Toxic megacolon | 1. Bold (+ 2) |
| | 2. What extra-intestinal manifestations occur in ulcerative colitis? | 2. Extra-intestinal Manifestations<br>2.1. Polyarthritis,<br>2.2. sacroiliitis, ankylosing spondylitis<br>2.3. Uveitis<br>2.4. Skin lesions<br>2.5. Pericholangitis<br>2.6. Primary sclerosing cholangitis | 2.<br>4 for pass |

Ulcerative Colitis 2005-2

**TOPIC:** Ulcerative colitis _____ **NUMBER: 4b**

| | | |
|---|---|---|
| **OPENING QUESTION** | What are the characteristic pathological features of Ulcerative colitis? | **COMMENTS** |
| **POINTS REQUIRED** | 1 Colon only | 4 of 7 for pass |
| | 2 Continuous ..no skip leasions | 40% |
| | 3 Chronic, relapsing | |
| | 4 Crypt abscesses | |
| | 5 Mucosal ulceration, with pseudopolyps | |
| | 6 Mucosal inflammation only | |
| | 7. Epithelial dysplasia | |
| | * Comparison of Crohns and UC acceptable | |
| **PROMPTS** | | |
| **SECOND QUESTION** (if needed) | What are the complications of UC? | 40% |
| **POINTS REQUIRED** | 1 Primary sclerosing cholangitis | 2 of 3 for pass |
| | 2 Toxic megacolon | |
| | 3 Neoplasia | |
| **PROMPTS** | What are the extra-intestinal manifestations of Crohn's Disease? | |
| **THIRD QUESTION** (if needed) | What are the features of dysplasia/ neoplasia in UC? | 20% |
| **POINTS REQUIRED** | 1 Often multi-focal | 1 of 2 for pass |
| | 2 Carcinomas are often infiltrative without obvious exophytic masses | |
| **PROMPTS** | | |