Digestive System 3

Lecture 13
Pathology and Clinical Science 1 (BIOC211)
Department of Bioscience

Text Reference:
SESSION LEARNING OBJECTIVES

- Understand the normal function lower tract small and large intestines
- Understand the causation, clinical features, treatment aims and prognosis for the following conditions
- Pancreas
  - acute and chronic pancreatitis
- Small intestine/ Large intestine
  - Inflammatory Bowel Disease,
  - Irritable Bowel syndrome
BE HEARD.

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Fill it out for a chance at winning $1000* and help us improve higher education.
The sooner you complete it, the more chances you have to win!

OVERVIEW SMALL INTESTINE

- **Small bowel function**
  - absorption carbohydrate (CHO), protein, lipids, calcium, B12, iron (Fe)

- **Small bowel disorder - clinical features**
  - diarrhoea,
  - abdominal pain - site
  - bloating
  - weight loss
  - nutritional deficiencies
# SMALL BOWEL INVESTIGATIONS

- Blood Full Blood Evaluation (FBE), proteins, calcium, B12
- Autoantibodies, endomysium, tissue transglutaminase, reticulin, gliadin
- Barium follow through
- Jejunal biopsy
- 3 day fat collection (100 gm fat intake)
- Lactose intolerance (intake of 50 gm lactose)
# PANCREATITIS

Inflammation and oedema of pancreatic tissue caused by presence of intracellular proteases, often initiated by high intracellular levels of Calcium, causing mild fat necrosis or severe necrosis and haemorrhage.

Can be acute or chronic.

**Cause** - majority 90%:

- gallstones causing reflux of bile into pancreatic duct so activating trypsin
- alcohol by stimulating secretion and blocking sphincter that releases enzymes into small intestine
- other causes (rare) idiopathic, infections, iatrogenic (post Endoscopic Retrograde Cholangiopancreatography (ERCP)), drugs, trauma
ACUTE PANCREATITIS

Clinical Features

- epigastric & abdominal pain increasing in severity over 15-60 minutes often following intake large meal or alcohol
- nausea & vomiting
- signs of shock
- low grade fever
- abdominal distention, tenderness, guarding,
- reduced or absent bowel sounds

Investigations

- serum amylase levels (x3), serum lipase
- FBE, ESR & C-Reactive protein
- plain X-ray, Ultrasound, contrast CT Scan
ACUTE PANCREATITIS

FIGURE 15-1
Pathogenesis of acute pancreatitis. Injury to the ductules or acinar cells leads to the release of pancreatic enzymes. Lipase and proteases destroy tissue, thereby causing acute pancreatitis. Release of amylase is the basis of a test for acute pancreatitis.

**FIGURE 15-2**

Acute hemorrhagic pancreatitis. (A) Large areas of the pancreas are intensely hemorrhagic. (B) The cut surface of the pancreas in a less severe case of acute pancreatitis, and at a somewhat later stage than in panel A, shows numerous, yellow-white foci of fat necrosis.

ACUTE PANCREATITIS

http://www.pathologyoutlines.com/images/pancreas/7_02.jpg
# ACUTE PANCREATITIS

## Treatment
- usually emergency
- no oral intake, but restoration of lost plasma via I.V route
- Analgesia
- oxygen therapy / gastric suction

## Complications - 10% mortality
- abscess formation, pseudocyst, necrotizing pancreatitis
- obstruction of head of pancreas or duodenum
- haemorrhage
- chemical peritonitis
- shock
- septicaemia
PANCREATIC ABSCESS

http://www.health-res.com/EX/07-31-12/2119.fig7.jpg
## CHRONIC PANCREATITIS

Chronic intra pancreatic enzyme activity leading to fibrosis

### Cause
- Alcohol - 60 - 80%, cystic fibrosis, inherited defect

### Clinical Features
- Episodic pain radiating to back
- Anorexia, weight loss, diarrhoea
- Signs of diabetes and malabsorption

### Investigations
- Ultrasound, contrast CT, Magnetic retrograde cholangiopancreatography (MRCP)

### Treatment
- NSAIDS, analgesia, tricyclics, oral pancreatic enzymes, low fat diet
CHRONIC PANCREATITIS

FIGURE 15-5
Chronic calcifying pancreatitis. (A) The pancreas is shrunken and fibrotic, and the dilated duct contains numerous stones. (B) Atrophic lobules of acinar cells are surrounded by dense, fibrous tissue infiltrated by lymphocytes. The pancreatic ducts are dilated and contain inspissated proteinaceous material.
CHRONIC PANCREATITIS

http://www.pathologyoutlines.com/images/pancreas/7_01.jpg
# INFLAMMATORY BOWEL DISEASE

<table>
<thead>
<tr>
<th>Crohn’s (CD) &amp; Ulcerative Colitis (UC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Inflammation of intestine - immune mediated</td>
</tr>
<tr>
<td>• Occurs more in western groups</td>
</tr>
<tr>
<td>• Male / female affected, slightly more female than male in Crohn’s</td>
</tr>
<tr>
<td>• Cause unknown but current findings suggest a genetic susceptibility with an environmental trigger</td>
</tr>
<tr>
<td>• Episodes of remission &amp; exacerbations</td>
</tr>
<tr>
<td>• UC has risk for colorectal cancer</td>
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**INFLAMMATORY BOWEL DISEASE**

Findings and Theories under research

<table>
<thead>
<tr>
<th><strong>Familial tendency</strong></th>
<th>Both CD and UC</th>
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</thead>
</table>
| **Dietary factors**   | Butyric acid, sulphides  
                            Omega 3 fatty acids |
| **Smoking**           | High in CD / ex-smokers UC |
| **Infective agents**  | Mycobacterium paratuberculosis, measles, yeasts, Listeria & Helicobacter species |
| **Familial tendency** | Both CD and UC |
| **Endogenous bacteria** | Suggestion bacteroides and strains of E Coli initiate & continues the inflammation |
| **Immunological factors** | Inability to down regulate immune response esp. to endogenous luminal antigens |
INFLAMMATORY BOWEL DISEASE

Crohn’s Disease

- Thickened narrowed small intestine
- Any area of GI tract, affected more commonly in terminal ileum, or (R) side of colon
- Initial ulceration of mucosal layer is affected then deepens to involve all layers (transmural) may develop to form fistulas, abscesses or adhesions
- Skip lesions
- Cobblestone appearance
- Granulomas present

Oesophageal Crohn’s

http://www.sciencephoto.com/image/390509/530wm/C0096124-Crohn’s_disease_in_the_oesophagus-SPL.jpg
FIGURE 13-31
Crohn’s disease. The mucosal surface of the colon displays a “cobblestone” appearance owing to linear ulcerations, as well as edema and inflammation of the intervening tissue.
CROHN’S DISEASE

CROHN’S DISEASE

B. Changes in the intestinal wall

Rigid, thickened wall (fibrosis)
Normal thin flexible wall
Inflammation
Narrow lumen leading to obstruction
Ulcers in mucosa

Area of acute inflammation and ulceration
Thick wall and narrow lumen of intestine

D. Fistula—abnormal opening between two structures

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INFLAMMATORY BOWEL DISEASE

### Ulcerative Colitis

- Spreads proximally
- May affect
  - rectum - proctitis
  - Sigmoid - proctosigmoiditis
  - Whole colon - pancolitis
- only mucosa and submucosa layers affected
- mucosa reddened, inflamed, bleeds easily
- inflammatory cells in lamina propria and crypts

![Ulcerative Colitis Image](http://www.sciencephoto.com/image/415157/530w/m/C0103581-Ulcerative_colitis-SPL.jpg)
ULCERATIVE COLITIS

FIGURE 13-34
Ulcerative colitis. Prominent erythema and ulceration of the colon begin in the ascending colon and are most severe in the rectosigmoid area.

Complications of Ulcerative Colitis

COMPARISONS

CROHN'S DISEASE
- Stricture
- Linear ulceration
- Fissures
- Skip lesions

Small intestine
Transmural inflammation

ULCERATIVE COLITIS
- Pseudopolyp
- Continuous colonic involvement, beginning in rectum
- Active disease: superficial ulceration
- Active disease: atrophy
- AND/OR

Large intestine
INFLAMMATORY BOWEL DISEASE

**Investigations**

- Stool cultures
- Barium follow through
- FBE ESR, Fe studies
- C reactive protein
- Colonoscopy not in acute stage
- Ultrasound
- Radionuclide scans
**IBD - CLINICAL FEATURES**

- **Crohn's Disease**
  - 15% have no GIT symptoms
  - wt loss
  - mouth ulcers
  - anaemia
  - diarrhea
  - cramping pain
  - R iliac fossa mass if colon
  - melaena
  - malabsorption picture

- **Ulcerative Colitis**
  - urgency and frequency of stool > 6 per day, may be as high as 20
  - watery bloody diarrhoea, mucus may be present
  - low cramping abdominal pain
## INFLAMMATORY BOWEL DISEASE

### Treatment

- **Anti inflammatory medications e.g.** glucocorticoids, amino salicylates - Sulphasalazine, immunosuppressants - azathioprine

- **Remove trigger factors e.g.** emotional and stress triggers

- **Nutritional supplements / I.V administration of** hypertonic glucose solutions with amino acids and fats.

- **Prevent complications**

- **Surgery – drainage of abscess/ repair of fistulas**
**IRRITABLE BOWEL SYNDROME**

A syndrome or collection of bowel symptoms present with **no** structural or biochemical abnormalities
(functional disorder)

- 1 in 5 persons have symptoms of IBS
- 50% seek professional medical advice, more female than men 3:2
- Non-GIT conditions may be present eg Chronic Fatigue Syndrome, Fibromyalgia, TMJ joint dysfunction
- Often stress related
IRRITABLE BOWEL SYNDROME

Clinical Features

• Persistent or recurring abdominal pain / discomfort / distension / bloating
• Altered bowel function
• Flatulence
• Constipation / diarrhoea
• Nausea & anorexia
• Anxiety or depression

Triggers include -

• GIT infection, antibiotic therapy, pelvic surgery , psychological stress
IRRITABLE BOWEL SYNDROME

Diagnostic Criteria

- 12 consecutive weeks of abdominal pain in 1 twelve month period
- To include 2 of the following 3 symptoms
  - Symptoms relieved by defecation
  - Onset change of frequency of stool
  - Onset change in form of stool

Treatment

- Organ Treatment,
  - Dietary triggers, fibre, anti-diarrhoeal, smooth muscle relaxants,
- Central Treatment
  - Physical explanation of symptoms
  - Counselling, psychotherapy, hypnotherapy, cognitive behavioural therapy, antidepressants
**IRRITABLE BOWEL SYNDROME**

<table>
<thead>
<tr>
<th>Treatment continued…..</th>
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<tr>
<td>• Increased fibre in diet except in acute stage</td>
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<tr>
<td>• Avoid dietary triggers (gas producing foods)</td>
</tr>
<tr>
<td>• Antispasmodic / anticholinergic drugs</td>
</tr>
<tr>
<td>• Alosetron – a 5-HT₃ antagonist</td>
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<tr>
<td>• Surgery – drainage of abscess/ repair of fistulas</td>
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## APPENDICITIS

### Epidemiology
- Extremely common
- 12% males and 25% females in USA

### Pathologenesis
- Related to intraluminal obstruction with: faecalith/ gallstone/ tumour/ parasites or lymphatic tissue
- Appendix becomes inflamed, swollen and gangrenous
- Perforates if not treated
# APPENDICITIS

## Clinical Features
- Sudden onset of referred epigastric or periumbilical pain.
- Pain becomes colicky and localised to lower (R) quadrant.
- Deep tenderness
- Spasm of abdominal muscles and guarding
- Nausea / vomiting

## Diagnosis / Investigations
- Patient history
- Ultrasound

## Complications
- Peritonitis / periappendiceal abscess formation/ septicemia
INFLAMMATION OF APPENDIX

(a) Normal appendix

(b) Inflamed appendix

http://biology-forums.com/gallery/14755_26_10_12_5_22_58_96792213.jpeg
INFLAMMATION OF APPENDIX

Appendix, Acute Appendicitis

red color c/w acute inflammation

http://www.iupui.edu/~pathol/c603_General/labs/Inflammation%20Lab/lab_images/720edb.jpg
Readings and Resources

Resources:

- **Set Textbooks:**

- **Additional textbooks:**
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