# 2.0 DISORDERS OF GASTROINTESTINAL TRACT

## 2.1 Peptic Ulcer Disease

The term peptic ulceration refers to an ulcer in the lower esophagus; stomach and duodenum. They have in common the involvement of acid-pepsin in their pathogenesis leading to disruption of the mucosal integrity causing local defect or excavation due to active inflammation. The common ulcers are duodenal and/or gastric.

Peptic ulcer may present in many different ways, the commonest is chronic, episodic pain present in many different ways, and may persist for months or years. However, the ulcer may come to attention as an acute episode with bleeding or perforation, with little or no previous history. As with duodenal ulcer, epigastric pain is the commonest symptom of gastric ulcer.

### 2.1.1 Gastro Esophageal Reflux Disease. (GERD)

It is a disorder resulting from gastric acid and other gastric contents into the esophagus due to incompetent barriers at the gastro esophageal junction.

**Diagnosis**

- Heartburn and regurgitation of sour material into the mouth are specific symptoms
- Symptoms for persistent disease may include odynophagia, dysphagia, weight loss and bleeding
- Extra esophageal manifestation are due to reflux of gastric contents into the pharynx, larynx, trachealbrochial tree, nose and mouth causing chronic cough, laryngitis, pharyngitis. It may also cause or aggravate chronic bronchitis, asthma, COPD, pneumonia, chronic sinusitis and dental decay.

**Investigation**

Diagnosis clinically by history alone and therapeutic trial of H2 receptor blocker or proton pump inhibitors (PPI) such as cimetidine 400mg 12hourly **or** Omeprazole 40mg 12 hourly respectively for 1 week, provides support for diagnosis of GERD. Esophagoscopy is valuable but not diagnostic for GERD, double contrast Barium meal is acceptable alternative to patient unwilling to undergo endoscopy. The 24-hours esophageal PH Metry is the specific procedure to confirm presence of GERD.

**Treatment**

The goals of treatment are to provide symptom relief, heal erosive esophagitis and prevent complication. Life style changes and antisecretory agents may be adequate.

**Drug of choice** is H2 Receptor blockers which are effective in symptoms relief and are considered as first line

**C:** Ranitidine 150mg (O) 12 hourly for 14 days;

Children 2 -4mg/kg 12 hourly for 14 days.

Proton Pump inhibitors (PPI) are considered as **second line** and are much more effective in healing ulcers or erosive esophagitis.

**Drug of Choice**

**C:**Omeprazole 20mg (O) once daily for 4 -8 weeks

Children 10 -20kg body weight 10mg once daily for 4-8 weeks.

**Alternatively**

**D:** Esomeprazole 40mg (O) once daily for 4-8 weeks, then 20mg once daily for maintenance to prevent relapse.

**Referral**

Refer to specialized centers for all cases with persistent symptoms and/or new complications despite appropriate treatment above.

**NOTE**

Specific lifestyle changes for patient advice may include

- Reduce spices, and avoid foods and fruits that exacerbate pain in individual patients
- Stop smoking and avoid alcohol
- Low consumption of coffee or tea
- Avoid carbonated drinks
- Avoid medicines such as non-steroidal anti-inflammatory agents (NSAIDS) aspirin, steroids.

### 2.1.2 Gastro duodenal Ulcers (PUD)

**General Management**

- Consider peptic ulcer general measures as above
- Referral to a specialist is recommended in presence of persistent symptoms or new onset complications
- Endoscopic biopsy to exclude malignancy in all refractory cases is mandatory
- Evaluation and treatment of *H. Pylori* associated infection is mandatory for effective treatment.

**Management of *Helicobacter pylori* infection**

Gastric infection with the bacterium *H.Pylori* accounts for majority of PUD. It also plays role in development of gastric mucosal – associated lymphoid tissue (MALT) Lymphoma and Gastric adenocarcinoma.

**Laboratory diagnosis**

- Perform stool antigen testing; the test should be repeated 3 months after therapy to confirm eradication
- Perform urea breath tests; the test require the patient to be off PPI therapy for 14 days and same days after eradication therapy

- Perform biopsy for urease test; more specific, helpful in cases where antibiotic sensitivity testing is required
- Serology confirms the exposure but not necessarily an active infection

**Treatment**

Triple therapy is indicated for complete eradication of the organism.

Omeprazole (PO) 20mg twice daily + Amoxicillin (PO)1g twice daily + Metronidazole (PO) 400mg twice daily for 7 days

OR

Lansoprazole (PO) 30mg twice daily + Clarithromycin (PO) 250mg twice + Tinidazole (PO) 500mg twice daily for 7 days.

# 2.2 Ulcer Related Conditions

## 2.2.1 Non-ulcer Dyspepsia (Functional Dyspepsia)

Defined as ≥ 3 months discomforting postprandial fullness, early satiety, and epigastric pain/burning in the absence of organic cause. Most patient follow a benign course, but small number of patients with *H.Pylori* infection or those on NSAIDs progress to ulcer formation. It is the cause of symptoms in more than 60% of patients with dyspepsia.

**Diagnosis**

Diagnosis clinically as above, plus endoscopic exclusion of esophagitis, peptic ulceration, or malignancy

**Treatment**

- Eradicate *H.Pylori* if present, if symptoms continue or recurs use H2RB or PPI on per demand basis to control symptoms.
- Use of Prokinetic agents such as Domperidone or Metoclopramide in short course of 2 to 8 weeks, shows beneficial effect at reducing dyspeptic symptoms.

  **D:**Domperidone (PO): Adults: 10 -20 mg 6-8 hourly daily taken 30minutes before meals; Children: (5- 12 Years) 5 -10mg 6-8 hourly

  **OR**

  **C:**Metoclopramide (PO): Adults: 10mg 8hourly daily

  Children: 0.5mg/kg/day in 3devided doses daily

  Counseling and reassurance are important.

## 2.2.2 Gastritis

Acute gastritis is a term covering a broad spectrum of entities that induce inflammatory changes in the gastric mucosa. The different etiologies share the same general clinical presentation. However, they differ in their unique histological characteristics. The inflammation may involve the entire stomach (e.g., pan gastritis) or a region of the stomach (e.g., antral gastritis). Acute gastritis can be broken down into 2 categories: erosive (e.g., superficial erosions, deep erosions, hemorrhagic erosions) and non erosive (generally caused by *Helicobacter pylori*).

Common etiologies includes certain drugs, alcohol, bacterial, viral, and fungal infections; acute stress, radiation, allergy and food poisoning, bile, ischemia, and direct trauma.

**Diagnosis**

- Symptoms may include nausea, vomiting, loss of appetite, belching, and bloating
- Occasionally, acute abdominal pain can be a presenting symptom
- Fever, chills, and hiccups also may be present

**Note**

- The diagnosis of acute gastritis may be suspected from the patient's history and can be confirmed histologically by biopsy specimens taken at endoscopy
- A number of laboratory tests may also be ordered depending on suspected etiology which may include Full hemogram, Liver and Renal functions test

**Treatment**

- Administer medical therapy as needed, depending on the cause and the pathological findings
- No specific therapy exists for acute gastritis, except for cases caused by *H pylori*
- Administer fluids and electrolytes as required, particularly if the patient is vomiting
- Discontinue the use of drugs known to cause gastritis (e.g., NSAIDs, alcohol)
- Consider short course use of Antacids, H2RB or PPI for relief of symptoms

## 2.3 Management of GI Bleeding

Acute gastro intestinal (GI) bleeding is common medical emergency resulting in significant morbidity and mortality. It can occur anywhere from mouth to anus; it is therefore subdivided into upper gastrointestinal bleeding(UGIB), anatomically above the ligament of Treitz; and lower gastrointestinal bleeding(LGIB), which is further subdivided to small bowel bleeding(middle GIB) and Colonic bleeding.

Causes for UGIB include, erosive ulcerative disease, esophagitis, portal varices and gastropathy, vascular ectasias, Mallory weiss tear and tumours. Causes for LGIB include, Diverticular disease, hemorrhoids, anal fissures, infectious and radiation colitis, inflammatory bowel disease, polyps, tumours, vascular ectasias and intussusceptions especially in children.

**Diagnostic guide**

Begin with an assessment of patient’s hemodynamic status (normal, orthostatic hypotension, or shock), while trying to localize the acute GIB through focused history and examination. Include the following in history, description of bleeding, duration and frequency, prior bleeding, cormobidities, medications, previous surgery, recent polypectomy or prior radiation.

Assess for the vital signs, stigmata of liver disease, abdominal tenderness, stool colour by rectal examination, nasogatric aspiration may show a positive gastric aspirate.

**Diagnostic procedures:**

Do baseline investigation, Full hemogram, Coagulopathy profile, liver and renal functions. Specifically, upper and lower endoscopy is appropriately indicated. While Tagged red cell scan and Angiography would be indicated for rapidly or obscure bleeding patients.

**Treatment guide**

**1. Pharmacological**

- Intravascular volume replacement should be restored with either ringers lactate or isotonic saline through large bore IV lines.
- Blood transfusion with packed red blood cells should immediately follow. Correct severe thrombocytopenia with packed platelet concentrates, while overt coagulopathy should be corrected with fresh frozen plasma, and Vitamin K S.C injection 5 -10 mg stat given to stable patients.
- Institute (IV) proton pump inhibitors e.g. Esomeprazole 40mg 12hrly. For 3-5 days, then oral therapy up to 6 weeks.
- Add Octreotide 50mcg (IV) stat then 50mcg 8hrly (IV) for 3-5 days specifically for varecial bleeding
- Add ciprofloxacin 400mg 12hrly (IV), or Metronidazole 500 8hrly (IV), or Ceftriaxone 1gm 12hrly (IV) for 3-7 days especially in varecial bleeding.

**2. Non Pharmacological**

- Endoscopy done within 24 hours could confirm diagnosis and provide sustained hemostasis control. Therapeutic modalities include variceal band ligation, Hemocliping, sclerotherapy, injectional tamponade therapy, thermocoagulation and angiographic embolization.

**3. Surgical Management**

- TIPS or shunt therapy is indicated in patients with esophageal varices who have failed pharmacologic and endoscopy therapy or those with bleeding gastric fundic varices.
- Surgical laparatomy for small bowel resection or colectomy is indicated as salvage therapy for small group of patients whom pharmacological, endescopic, and angiotherapy have failed.

**Note:** Refer stabilized patients with GIB to specialized centres for expertise management.

## Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is an idiopathic disease, probably involving an immune reaction of the body to its own intestinal tract. The 2 major types of IBD are ulcerative colitis (UC) and Crohn disease (CD). As the name suggests, ulcerative colitis is limited to the colon. Crohn disease can involve any segment of the gastrointestinal tract from the mouth to the anus

### 2.4.1 Ulcerative colitis

Inflammatory condition that affects the rectum extends proximally to affect a variable amount of the colon. Smoking appears to worsen the disease condition.

**Diagnosis**

- Active disease is associated with diarrhea, rectal bleeding, tenesmus, passage of mucus, and crampy abdominal pain
- Severity of symptoms correlates with the extent of disease
- Occasionally, diarrhea and bleeding is intermittent and mild that the patient may not seek medical attention, thus though UC can present acutely, symptoms usually have been present for weeks to month
- Complication may present as, Massive hemorrhage (<1%); Toxic megacolon, Perforation with features of peritonitis, stricture.

**Note**

Diagnosis relies upon the patient's history; clinical symptoms; negative stool examination for bacteria, C.difficile toxin, ova and parasites; sigmoidoscopic appearance; and histology of rectal or colonic biopsy specimen. Single contrast barium enema alternative to sigmoidoscopy but is limited by biopsy access. Supportive laboratory test: CBC for anemia; Thrombocytosis, leucocytosis may reflect active disease.

**Treatment and Referral**

- Refer patients to specialized centers once disease is suspected for expertise management
- Cure is not available, goals of therapy are to induce and maintain remission

Drug of Choice

**C:** Sulphasalazine (PO): Adults, 1 gram four times a day for acute disease, reducing to 500mg four times a day for maintenance; Children over 2 years for acute attack use 40-60mg/kg body weight daily.

Maintenance dose 20-30mg/kg body weight daily.

**Plus**

**B:** Prednisolone (PO) 30-60mg once daily for severe, acute and extensive disease; reduces gradually according to disease severity.

**Note**

- Correction of fluid deficit and/or blood is important in acute severe forms which may necessitates hospitalization
- Nutritional therapy should target to replenish specific nutrient deficits
- Life long surveillance is required due to risk of bowel cancer
- Use steroids only when the disease is confirmed, to avoid exacerbation of existing illness.

### 2.4.2 Crohn's Disease

Crohn's disease is an idiopathic, chronic, transmural inflammatory process of the bowel that often leads to fibrosis and obstructive symptoms and can affect any part of the gastrointestinal tract from the mouth to the anus.

**Diagnosis**

- Mainly abdominal pain and diarrhea; weight loss, anorexia, and fever may be seen
- Growth retardation in children
- Gross rectal bleeding or acute hemorrhage is uncommon
- Anemia is a common complication due to illeal disease involvement
- Small bowel obstruction, due to stricturing
- Perianal disease associated with fistulization
- Gastroduodenal involvement may be mistaken for *H.Pylori* negative PUD

**Diagnostic consideration**

- Endoscopy gold standard for diagnosing colonic and terminal illeal disease and readily permits mucosal biopsy and balloon dilatation of any stricture
- Barium follow through is still standard method for evaluating the small bowel, though capsule enterescopy is superior
- Discriminating features that favours Crohn's from Ulcerative colitis include small bowel disease, mainly right sided colonic disease, rectal sparing, fistulization, and granulomas. Immunological makers: pANCA is predictive in 70% of UC but only15% in CD; Antibodies to *Saccharomyces cervisiae* are found in up to 50% CD and less often in UC. When done together specificity is further improved
- Supportive laboratory tests: CBC for anemia; thrombocytosis, leucocytosis, as serrogate sign of inflammation, iron and folate studies, liver functions test, electrolytes/micronutrient deficiency assessment (calcium, magnesium, zinc).

**Treatment**

- Refer suspected cases to specialized centers for expertise management
- Baseline management as for Ulcerative Colitis above

### 2.4.3 Pseudomembrenous colitis

*Clostridium difficile* is organism responsible for an infectious colitis that affects 1 of every 200 patients who are admitted to the hospital. Increasingly implicated as a significant cause of morbidity and mortality among hospitalized patients, *C difficile* colitis should also be recognized

among outpatient populations. Prior antibiotic exposure remains the most significant risk factor for development of disease. Antibiotics first seen with clindamycin, but amoxylin and the cephalosporin's are now most frequently implicated. Extreme age, recent GI surgery, malignancy, prolonged hospital stay are other risk factors.

**Diagnosis**

- Diarrhea and abdominal cramps occurs during first week, but can be delayed up to six weeks
- Nausea, fever, dehydration can accompany severe colitis
- Abdominal examination may reveal distension and tenderness.

**Note**

- Stool examination is sensitive on anaerobic culture facilities which reveals toxigenic and non toxigenic strains
- Enzyme immunoassays are available for toxins A and B in stool
- Sigmoidoscopy is highly specific if lesion is seen but insensitive compared to the above.

**Treatment**

Drug of choice

**A:** Metronidazole (PO): Adults, 400mg 8hourly for 5-days

Children 1 month-12 years: 7.5 mg/kg (max. 400mg) every 8 hours

Second line

**D:**Vancomycin (PO/IV): Adults, 125mg – 500mg 6hourly for 5- 10days

Children > 1month : 40mg/kg/day in devided doses.

## 2.5 Irritable Bowel syndrome

Irritable bowel syndrome (IBS) is a functional GI disorder characterized by abdominal pain and altered bowel habits in the absence of specific and unique organic pathology.

**Diagnosis**

- Abdominal discomfort of at least 3 months duration
- Bloating or feeling of distension
- Altered bowel habits (constipation and/or diarrhea)
- Exacerbations triggered by life events.
- Coexistance of anxiety and depression.

**Diagnostic Considerations**

- Hematology and biochemistry studies
- Stool microscopy
- Colonoscopy with biopsy

**Treatment**

- Refer patients to specialized centers for proper evaluation and management.
- Supportive therapies;
- Reassurance and explanation are essential.
- High fibre diet and eating a healthy diet.
- Relief of pain due to abdominal cramps

  **A:** Hyoscine butyl bromide 20mg (O) four times a day
- Relief of anxiety that may be making symptoms worse

  **C:** Diazepam 5-10 mg (O) 8 hourly

  Give short and infrequent courses only, in order to avoid dependance.
- If constipation is predominant in IBS encourage high fiber diet.
- If diarrhea predominant in IBS

  **C:** Loperamide 4mg (O) stat, followed by 2mg after each unformed stool until diarrhoea is controlled.
- Explore psycho-social factors in resistant cases and counseling.

## 2.6 Malabsorption syndrome

Malabsorption is a clinical term that encompasses defects occurring during the digestion and absorption of food nutrients by and infections of the gastrointestinal tract. Although presenting symptoms, such as diarrhea and weight loss may be common, the specific causes of malabsorption are usually established based on physiologic evaluations. The treatment often depends on the establishment of a definitive etiology for malabsorption. Etiologic examples include pancreatic insufficiency, bacterial overgrowth, celiac disease, tropical sprue, lactase deficiency, diabetic enteropathy, thyroid disease, radiation enteritis, gastrectomy and extensive small bowel resection.

**Diagnosis**

Depending on etiology, presentation may collectively include:

- Diarrhoea a commonest symptom which is frequently watery
- Steatorrhea due to fat malabsorption; characterized, by the passage of pale, bulky, and malodorous stools. Stools often float on top of the toilet water and are difficult to flush
- Weight loss and fatigue
- Flatulence and abdominal distention
- Edema due to hypoalbuminemia, and with severe protein depletion ascites may develop
- Anemias which can either be microcytic iron deficiency (celiac disease) or macrocytic vitamin B-12 deficiency (chrohn's disease or illeal resection).
- Bleeding disorders (Ecchymosis, melena, and hematuria) due to vitamin K malabsorption and subsequent hypoprothrombinemia.
- Metabolic defects of bones (osteopenia or osteomalacia) due to vitamin D deficiency. Bone pain and pathologic fractures may be observed. Malabsorption of calcium can lead to secondary hyperparathyroidism.

- Neurologic manifestations: Electrolyte disturbances, such as hypocalcaemia and hypomagnesaemia, can lead to tetany. Vitamin malabsorption can cause generalized motor weakness (pantothenic acid, vitamin D) or peripheral neuropathy (thiamine), a sense of loss for vibration and position (cobalamin), night blindness (vitamin A), and seizures (biotin).

**Treatment**

- Patients should be referred to specialized centers for proper evaluation and definitive management
- Two basic principles underlie the management of patients with malabsorption, as follows:
    - The correction of nutritional deficiencies
    - When possible, the treatment of causative diseases
- Nutritional support
    - Supplementing various minerals, such as calcium, magnesium, iron, and vitamins, which may be deficient in malabsorption, is important
    - Caloric and protein replacement also is essential
    - Medium-chain triglycerides can be used as fat substitutes because they do not require micelle formation for absorption and their route of transport is portal rather than lymphatic
    - In severe intestinal disease, such as massive resection and extensive regional enteritis, parenteral nutrition may become necessary.
- Treatment of causative diseases
    - A gluten-free diet helps treat celiac disease
    - A lactose-free diet helps correct lactose intolerance; supplementing the first bite of milk-containing food products with Lactaid also helps
    - Protease and lipase supplements are the therapy for pancreatic insufficiency
    - Antibiotics are the therapy for bacterial overgrowth
    - Corticosteroids, anti-inflammatory agents, such as mesalamine, and other therapies are used to treat regional enteritis.

## 2.7 Pancreatitis

Pancreatitis is an inflammatory process in which pancreatic enzymes auto digest the gland. It may present as acute pancreatitis, in which the pancreas can sometimes heal without any impairment of function or any morphologic changes, or as chronic pancreatitis, in which individuals suffer recurrent, intermittent attacks that contribute to the functional and morphologic loss of the gland.

### 2.7.1 Acute Pancreatitis

It is due to sudden inflammation of the pancreas due to pancreatic enzymes auto digestion. Common risk factors which trigger the acute episode are presence of gallstones and alcohol intake.

**Diagnosis**

● Severe, unremitting epigastric pain, radiating to the back
● Nausea and vomiting

● Signs of shock may be present
● Ileus is also common
● Local complications: inflammatory mass, obstructive jaundice, gastric outlet obstruction
● Systemic complication: sepsis, acute respiratory distress syndrome, acute renal failure

**Diagnostic considerations**

- Serum amylase, in counts over 1000U/L, but poor correlates with disease severity. Serum Lipase twice the normal limit has superior sensitivity and specificity.
- Complete blood counts, Urea and electrolytes, bicarbonate levels, liver transaminases and albumin, LDH, glucose, calcium, CRP, and lipid profile for modified Glasgow criteria to disease severity and outcomes.
- Abdominal ultrasound, Plain abdominal X-ray, Chest X-ray, CT Abdomen.

**Treatment**

- Prompt referral to specialized centers with intensive care facilities is recommended
- Principles of management include expertise supportive therapy:
    - Nil per oral regimen for few days up to weeks is indicated depending on severity.
    - Intravascular volume expansion (colloids/crystalloid)
    - Opiates analgesia and antiemetics usually required.
    - Prophylactic antibiotics in severe state, useful when there is evidence of sepsis(IV) ceftriaxone 1g 12hrly + Metronidazole 500mg 8hrly or Meronem 1g 8hrly
    - ERCP + Sphincterotomy when gallstones are present in the CBD.

### 2.7.2 Chronic Pancreatitis

Chronic pancreatitis is long-term (chronic) inflammation of the pancreas that leads to permanent damage. The most common cause for such a condition is long-term excessive alcohol consumption.

**Diagnosis**

- The most common symptom is upper abdominal pain that may be accompanied by nausea, vomiting and loss of appetite
- As the disease gets worse and more of the pancreas is destroyed, pain may actually become less severe
- During an attack, the pain often is made worse by drinking alcohol or eating a large meal high in fats.
- Because a damaged pancreas can't produce important digestive enzymes, people with chronic pancreatitis may develop problems with digesting and absorbing food and nutrients. This can lead to weight loss, vitamin deficiencies, diarrhea and greasy, foul-smelling stools.
- Over time, a damaged pancreas also can fail to produce enough insulin, which results in Diabetes.

**Diagnostic Consideration:**

● Abdominal X-ray, for evidence of pancreatic calcifications
● CT, MRCP, ERCP, and Endoscopic ultrasound are complementary
● Biochemical; Glucose tolerance test, serum vitamins (ADEK), hemoglobin and calcium levels,
●Pancreatic function tests: Secretin /CCK – secretory test, fecal elastese1 concentrations

**Treatment**
Referral is recommended for expertise evaluation and management in specialized centers. Because chronic pancreatitis cannot be cured, direct the treatment towards:

- **Relieving pain with pain-killers**- In rare cases, surgery/ ERCP to open blocked ducts or remove part of the pancreas may be done to relieve pain.
- **Improving food absorption** - The patient should be recommended to follow a low-carbohydrate, high-protein diet that also restricts some types of fats. Once digestive problems are treated, patient will usually gain back weight and diarrhea improves. Another way is by giving the patient pancreatic supplements containing digestive enzymes.
- **Treating diabetes** - Treat diabetes with careful attention to diet to help keep blood sugar levels stable. In some people, insulin injections and other diabetic medications are needed.

## 2.8 Peritonitis

Refers to inflammation of the peritoneum; it may be localized or diffuse in location, acute or chronic in natural history, infectious or aseptic in pathogenesis. Acute peritonitis is most often infectious usually related to a perforated viscus (secondary peritonitis); primary or spontaneous peritonitis refers to when no intraabdominal source is identified. Acute peritonitis is associated with decreased intestinal motility, resulting in distention of the intestinal lumen with gas and fluid. The accumulation of fluid in the bowel together with the lack of oral intake leads to rapid intravascular depletion with effects on cardiac, renal, and other systems.

Chronic peritonitis refers to longstanding inflammation of the peritoneum. Causes include repeated attacks of infection such as from pelvic inflammatory disease (PID), Metastatic lesions or foreign substances that induce inflammation, and chronic infections within the abdomen such as Tuberculosis.

**Diagnosis**

- Acute peritonitis is usually characterized by acute abdominal pain and tenderness, dehydration, fever, hypotension, nausea and vomiting and tachycardia.
- Complications include abscess formation, oliguria and shock.
- Similar features may be seen in spontaneous bacterial peritonitis (SBP), which occurs in cirrhotic patients with ascites. Bacterial translocation, bacteraemia and impaired antimicrobial activity contribute to its development. Gram negative bacilli (E. coli) commonly are a causative microbe.

**Diagnostic considerations: (specific)**

- Peritoneal fluid analysis for microscopy, microbiology, culture and sensitivity
- Macroscopic evaluation of the peritoneal fluid will exclude hemoperitoneum in trauma cases
- Blood cultures due to bacterimia
- Scanning procedures (ultrasound and/or CT scan) facilitates the diagnosis, Abdominal having the highest diagnostic yield.

**Treatment considerations**
Surgery remains a cornerstone of peritonitis treatment.

Antimicrobial therapy is adjunctive to surgical correction of underlying lesion or process and treatment will depend on causative agent.
Where cause is not known antibiotics of choice are:

**C:** Ampicillin (I.V) 1g every 6hours for 5-10 days

**Plus**

**A**: Gentamicin (I.V) 4 mg/kg/24 hours in 3 divided doses for 5-10 days

**Plus**

**C:** Metronidazole (I.V)/ (O) 400-600mg every 8 hours for 5-10 days.

**Referral**

- Patient needs referral to centers where surgical intervention is adequate (i.e. expertise and medical facility)
- Refer to TB section for TB peritonitis management.

## 2.9 Constipation

According to the Rome III criteria for constipation, a patient must have experienced at least 2 of the following symptoms over the preceding 3 months: Fewer than 3 bowel movements per week; straining; lumpy or hard stools; sensation of anorectal obstruction; sensation of incomplete defecation, manual maneuvering required defecating.

Constipation is a symptom, not a disease. Contributory factors may include inactivity, low fiber diet and inadequate water intake. Specific causes may include, conditions associated with neurologic dysfunction, scleroderma, drugs, hypothyroidism, hypokalemia, hypercalcemia, Cushing's syndrome, colonic tumours, anorectal pain, and psychological factors.

**Diagnosis**

- Fewer than three bowel movements per week, small, hard, dry stools that is difficult or painful to pass, need to strain excessively to have a bowel movement, frequent use of enemas, laxatives or suppositories are characteristic.
- Other features may include; abdominal bloating, rectal bleeding, spurious diarrhea, low back pain, feeling of incomplete evacuation, and tenesmus.

**Referra**l
The following signs and symptoms, if present, are grounds for urgent evaluation or referral:

- Rectal bleeding
- Abdominal pain
- Inability to pass flatus
- Vomiting
- Unexplained weight loss.

**Diagnostic guides:**
An extensive work up of the constipated patient is performed on an outpatient basis and usually occurs after approximately 3-6 months of failed medical management. It is advised to refer the patient at this juncture to specialized centers.