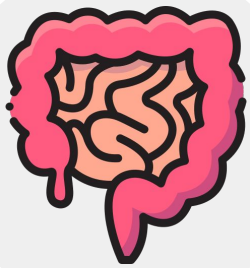
Abdominal pain and IBS



Objectives :

- ★ **Abdominal pain includes:**
  - ○ **PUD**
  - ○ **Acute pancreatitis**
  - ○ **Celiac disease** (covered in diarrhea lecture)
- ★ **Causes of Abdominal pain**
- ★ **Functional dyspepsia**
- ★ **Approach to management of dyspepsia**
- ★ **Management of H pylori**
- ★ **Irritable bowel syndrome**

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Introduction to Acute abdominal pain

Introduction

- **Abdominal pain** accounts for approximately 50% of all urgent admissions to general surgical units.
- There are four types of abdominal pain:

Visceral

- Visceral Peritoneum Lining the organs, nerve endings are mostly all **autonomic**
- Gut organs are insensitive to stimuli such as burning and cutting but are sensitive to distension, contraction, twisting and stretching.
- Pain from unpaired structures is usually, but not always, felt in the midline.

Parietal

- Parietal Peritoneum Lining the sides toward the abdominal wall. Here we have muscle nerves passing **(somatic)**
- The parietal peritoneum is innervated by somatic nerves and its involvement by inflammation, infection or neoplasia causes sharp, well-localised and lateralized pain.

Referred pain

- Pain perceived at a site distant from the source of stimulus why? The theory is based on the embryological origin of the organs & interconnected nerves
- Gallbladder pain, for example, may be referred to the back or shoulder tip.
- Right Shoulder Pain —> With Cholecystitis and Perforated PUD
- Left Shoulder Pain —> With Diaphragmatic irritation due to splenic rupture
- Left-Sided Chest and Arm Pain —> MI

Psychogenic

- Cultural, emotional and psychosocial factors influence everyone's experience of pain.
- In some patients, no organic cause can be found despite investigation, and psychogenic causes (depression or somatization disorder) may be responsible

- **The acute abdomen is a consequence of one or more pathological processes:**

Inflammation

- Pain develops **gradually**, usually over several hours.

Perforation

- Pain starts **abruptly**; it is severe and leads to **generalised peritonitis.**

Obstruction

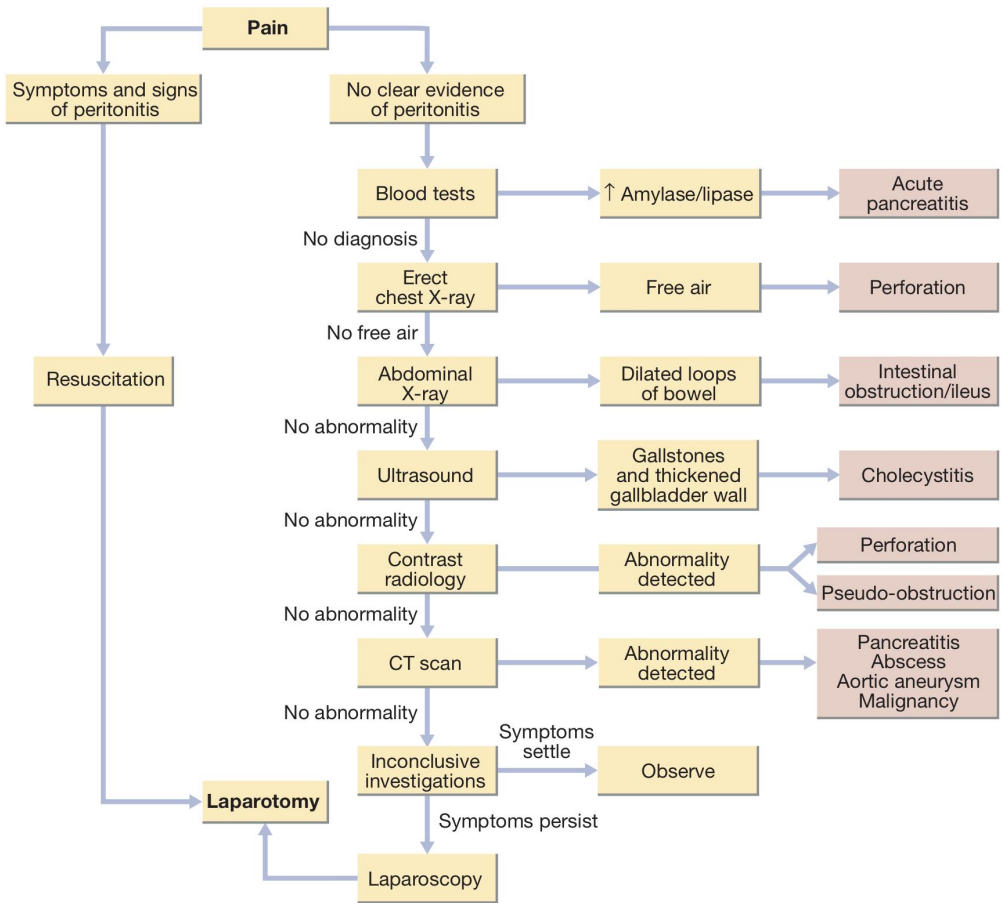
- Pain is **colicky**, **with spasms** that cause the patient to writhe around and double up.

Introduction to Acute abdominal pain

◀ Causes of acute abdominal pain

Surgical	Medical
• Appendicitis • Cholecystitis • Bowel obstruction • Acute mesenteric ischemia • Perforation • Trauma • Peritonitis	• Cholangitis (inflammation of bile duct) • Pancreatitis • Choledocholithiasis (stone in the bile duct) • Diverticulitis • PUD Peptic Ulcer Disease • Gastroenteritis • Non-abdominal causes that can cause abdominal pain as MI in diabetic and HTN patients • **FUNCTIONAL causes (More of chronic)**[1]

◀ Approach to Acute abdominal pain



1- Our talk is going to be about functional causes which are chronic pain. **Acute pain is more serious than chronic pain**. why? Because chronic pain is less likely to be an inflammation or cancer. **Most abdominal pain that requires surgery is acute**, less likely chronic abdominal pain needs surgery e.g. bowel ischemia "bowel angina"

Introduction to Dyspepsia

Definition

- **Dyspepsia:** is a collection of symptoms including nausea bloating and epigastric pain
- **Functional Dyspepsia (non-ulcer dyspepsia):** This is defined as chronic dyspepsia in the absence of organic disease.

Definition: (Rome III committee):

There is a new classification including two distinct diagnostic categories "for functional dyspepsia":

- ★ **Postprandial distress syndrome (PDS)** (After meals, fullness,heaviness , feeling nausea, bloating or early satiety, there is more of discomfort rather than a pain)
- **Epigastric pain syndrome (EPS)** which indicate meal-related and unrelated symptoms (Meal-related epigastric **pain** and unrelated epigastric pain)

◄ Etiology

- Most common cause of epigastric pain is **functional[1] dyspepsia (60%)**.
- Abdominal wall pain is worsened by movement and may cause guarding

Dyspepsia caused by structural[2] or biochemical causes

- Peptic ulcer, GERD, Biliary pain, Chronic abdominal pain, Gastric/esophageal cancer.
- Gastroparesis, pancreatitis, Carbohydrate malabsorption
- Medications (Including K+ supplements, digitalis, iron, theophylline, oral antibiotics (especially ampicillin and erythromycin), NSAIDs, corticosteroids, niacin, gemfibrozil, narcotics, colchicine quinidine, estrogens, levodopa)
- Infiltrative disease of the stomach (e.g. Crohn's disease sarcoidosis)
- Metabolic disturbances (hypercalcemia, hyperkalemia)
- Hepatoma, Ischemic bowel disease
- Systemic disorders (DM, Thyroid and parathyroid disorders, connective tissue disease)
- Intestinal parasites (Giardia, strongyloides)
- Abdominal cancer, especially pancreatic cancer

◄ Clinical approach

History:

First step is to rule out organic causes of dyspepsia and that's done by history.

- Ulcer-like or acid dyspepsia (eg, burning, epigastric hunger pain with food, antacid, and antisecretory agent relief).
- Dysmotility-like dyspepsia (with predominant nausea, bloating, and anorexia).
- Unspecified dyspepsia.

Physical examination

Second step

- **Usually normal**
- Presence of palpable mass needs further action

1) Functional dyspepsia is structurally/organically normal, usually related to motility (spams) or altered perception of pain
2) Structural abnormalities is also called organic causes

Functional dyspepsia

◀ Alarm symptoms [1]: ★

VAW GOt a PhD

Unintended Weight loss	**Persistent Vomiting**	**Progressive Dysphagia**	**Odynophagia** (Pain with swallowing)	**unexplained Anemia or iron deficiency**
Hematemesis, Melena	**Palpable abdominal mass or lymphadenopathy**	**Family history of upper gastrointestinal cancer**	**Previous Gastric surgery**	**Jaundice / Lymphadenopathy / mass / Ascites**

NPV=99%: **Negative Predictive Value of 99.%** means if all of the above is negative, then the chance that this patient has "functional dyspepsia" is 99%. So having one of these symptoms denotes organic causes.

◀ Investigations:

Routine laboratory tests [2]

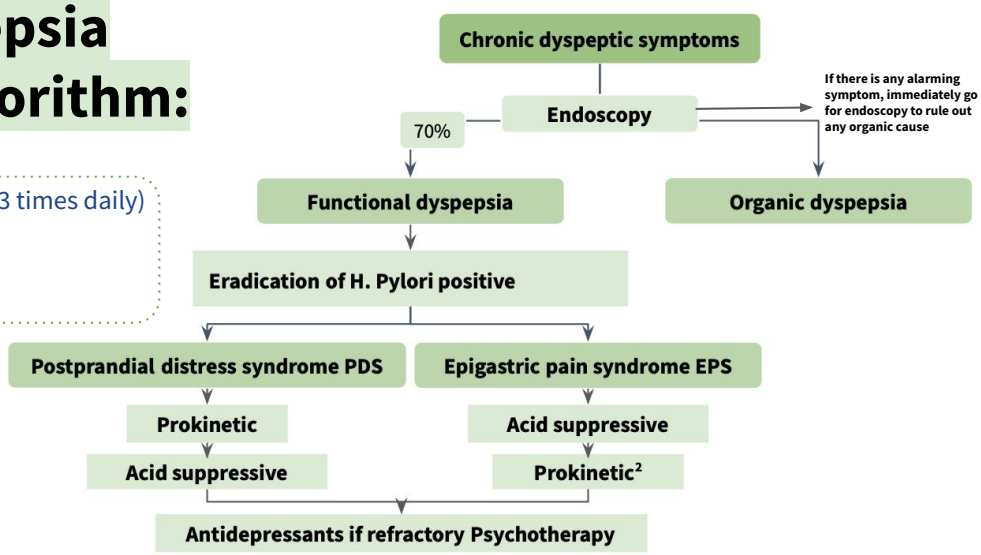
- **We usually start with basic workup like Routine blood counts and blood chemistry:** LFTs, RFTs To rule out any extra-intestinal cause of abdominal pain; Anemia, diabetes and hyperthyroidism .. etc"

Endoscopy (GOLD standard)

- **Gold standard test to exclude gastroduodenal ulcers, reflux esophagitis, and upper gastrointestinal cancers.**
- Beneficial because up to **40%** of patients have an organic cause of dyspepsia.
- It also provides reassurance to patients.
- Disadvantages:
  - Expensive, Invasive
  - Not cost-effective in young patients without alarm symptoms as up to **50%** -60% are normal.

◀ Functional dyspepsia management algorithm:

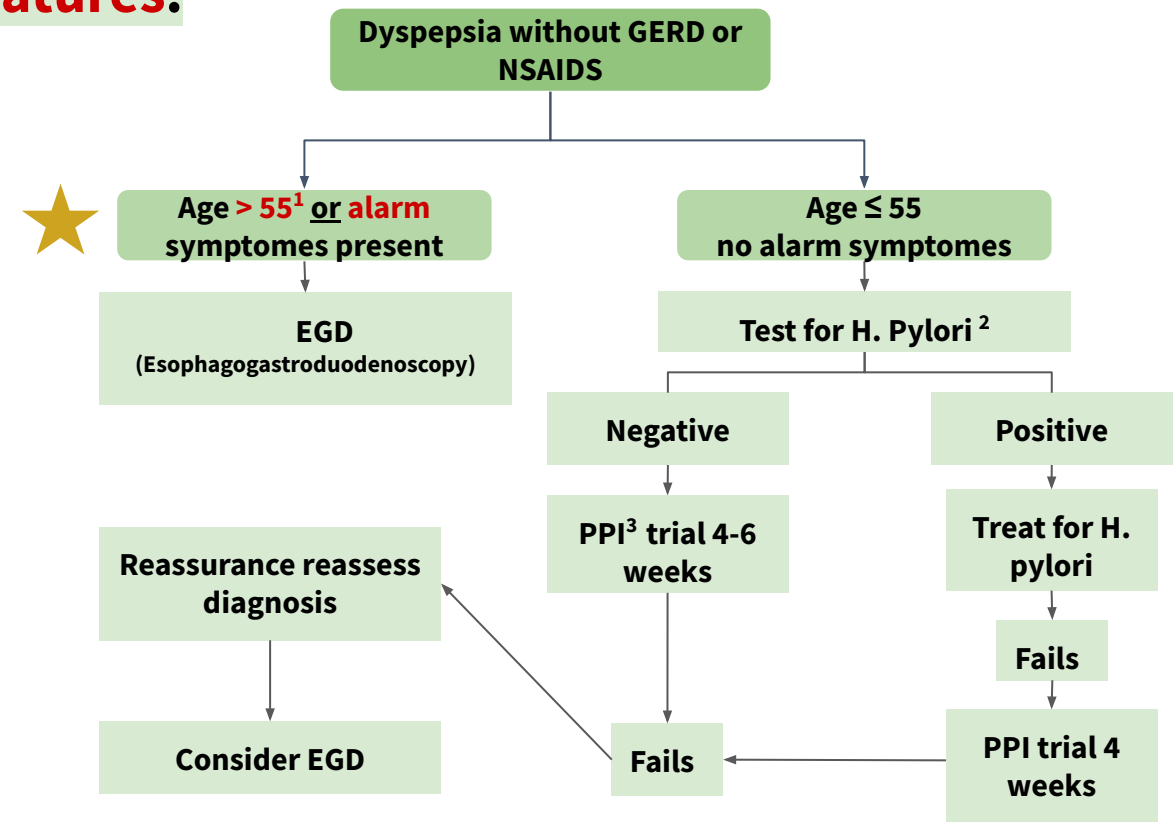
Prokinetic: such as metoclopramide (10 mg 3 times daily) or domperidone (10–20 mg 3 times daily)
Antacids: such as hydrotalcite
Antidepressants: such as Amitriptyline



1) Go through it anatomically: 1- Esophagus (Dysphagia, vomiting) 2- Stomach (Loss of weight, Loss of appetite, Hematemesis) etc.
2) It is always third step unless invasive there is indication

Functional dyspepsia

◄ Management of dyspepsia based on age and alarm features:



◄ Management of non responding functional dyspepsia

H. pylori negative functional dyspepsia (normal endoscopy) and failed and adequate trial of PPI

1- Re-evaluate the symptoms and diagnosis
2- Consider other sources of abdominal pain: pancreas, colon, biliary tract
3- Does the patient have symptoms or delayed gastric emptying? (Like gastroparesis in diabetics)
4- Does the patient have IBS?
5- Does the patient have panic disorder or other psychological issued?

Persistent symptomes no other cause established

Consider: Antidepressants, hypnotherapy, behavior therapy, prokinetic agents

Click here for a nice algorithm

1) So if a **60** yo presented with symptoms of functional dyspepsia, go with ENDOSCOPY
2) Highly prevalent cause dyspepsia. especially with Functional dyspepsia (absence of organic causes)
3) Proton pump inhibitors, improve the pain and burning

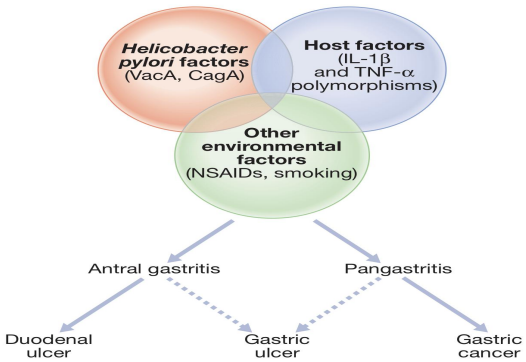
Peptic Ulcer Disease (PUD)

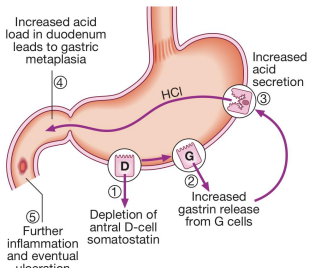
Definition

- The term 'peptic ulcer' refers to an ulcer in the lower oesophagus, **stomach** or **duodenum**, in the jejunum after surgical anastomosis to the stomach or, rarely, in the ileum adjacent to a Meckel's diverticulum.
- **What's the difference between an ulcer and erosion?**
  - Ulcers: Penetrate muscularis mucosae
  - Erosions: Don not penetrate muscularis mucosae
- Note: Gastric and duodenal ulcers coexist in 10% of patients and more than one peptic ulcer is found in 10–15% of patients.

◄ Etiology:

1) **Helicobacter pylori infection 80%[1] (most common)***
2) **NSAIDs (e.g. Aspirin):** inhibit prostaglandin production which is the major stimulant for mucus production that form the protective barrier, leads to impaired mucosal defenses.
3) **Zollinger–Ellison syndrome**
4) **Smoking (**Gastric more than duodenal)

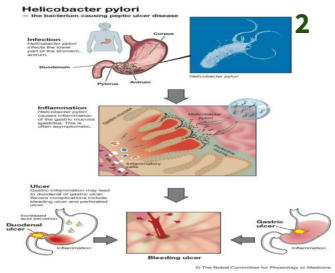


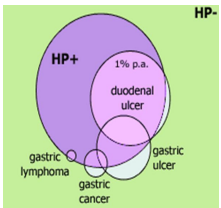


***The strongest evidence for the pathogenic role of H. pylori in peptic ulcer disease is the marked decrease in the recurrence rate of ulcers following the eradication of infection.**

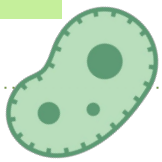
◄ Epidemiology & Transmission of H. pylori infection:

- **Socioeconomic status of the family is the main risk factor** as reflected by the level of sanitation and household hygiene. These infections are **probably acquired in childhood** by **person-to-person** contact (Fecal-oral route, Gastro-oral route or Oral-oral route)
- The vast **majority of colonised people remain healthy and asymptomatic**, and only a minority develop clinical disease.
- **95% of duodenal ulcer** patients and **85% of gastric ulcer** patients **are infected with H. pylori**. The remaining 15% of gastric ulcers are caused by NSAIDs.





◄ Microbiology of H. pylori:



❖ **Gram <u>negative spiral</u> organism with following characteristics:**
- Slow growing, **microaerophilic**
- Has multiple flagella at one end, which make it **highly motile**, allowing it to burrow and live beneath the mucus layer adherent to the epithelial surface.
- **Urease producing**

1) The most common human infection worldwide
2) Once H. pylori acquired it causes mucosal inflammation (Gastritis) that is often asymptomatic which leads to ulcer

Peptic Ulcer Disease (PUD) *cont'*

◄ Clinical presentation:

- Recurrent episodes of **epigastric pain** that is described as **dull**, sore, and gnawing. Although the most common cause of upper GI bleeding is PUD, the majority of those with ulcers do not bleed.
- **How to differentiate between gastric and duodenal ulcer?**
  - **Gastric ulcer:** pain is **G**reater after a meal, hence the weight loss.
  - **Duodenal ulcer:** pain **D**ecreases after a meal

EXTRA	Duodenal ulcer	Gastric ulcer
Pathogenesis	Caused by an increase in offensive factors (higher rates of basal and stimulated gastric acid secretion)	Caused by a decrease in defensive factors (gastric acid level is normal/low unless ulcer is pyloric or prepyloric)
H.pylori infection	90%	85%
Malignant potential	**Low** (malignancy is very rare)	**High** (5%-10% are malignant); therefore must **always be biopsied** and followed up to ensure healing.
Locations	Majority are 1-2 cm distal to pylorus (usually on posterior wall)	Type **I** (Most common): on **less**er curvature Type **II**: Gastric and duodenal ulcer **(Two)** Type **III**: **Pre**pyloric (within 2cm of pylorus) Type **IV**: Near esophagogastric junction **(Door)**
Age distribution	Occurs in younger patients (<40 y/o)	Occurs in older patients (>40 y/o)
Associated blood type	Type O	Type A
Risk factors	NSAIDs	Smoking
Other	- Eating usually relieves pain - Nocturnal pain is more common than in gastric	- Eating worsens pain - Complication rates are higher than those of duodenal ulcers. There is a higher recurrence rate with medical therapy alone

One is **Less**, **Two** has **Two**, **Three** is **pre**, **Four** is by the **door**

◄ Investigations:

1) Endoscopic biopsy is the **gold standard**
2) If **perforation** is suspected, perform upright **CXR** to evaluate air under the diaphragm or CT scan of the abdomen.
3) In **recurrent or refractory cases**, check serum **gastrin levels** to screen for Zollinger-Ellison syndrome.
4) Patients should be tested for H. pylori infection (discussed in next page)

Peptic Ulcer Disease (PUD) *cont'*

◀ Tests for H.pylori:

Non-invasive

1) **Serum antigen:** Detects IgG antibodies to H.pylori. Lacks specificity as it can't differentiate between active and treated disease. (Low diagnostic yield)
2) **Urea Breath Test (UBT):** H. pylori urease converts radiolabeled urea (C14 or C13) to CO2 and ammonia; this test detects CO2 formed from urea metabolism. Has high sensitivity and specificity. PPIs may cause false results.
3) **Stool antigen:** Detects H. pylori antigens in stool. Cost-effective initial test for H.pylori. Patients should be off PPIs for 2 weeks.

Both of **UBT** and **stool antigen** methods are good to measure the **effect of treatment**

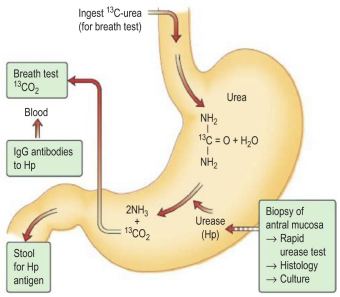


Figure 13.21 **Metabolism of urea by *Helicobacter pylori* (Hp).** The different tests available for the detection of *H. pylori* are shown.

Invasive (Endoscopic)

1) **Histology:** Specific but has a lot of false negatives (usually due to PPIs). H. pylori can be detected histologically on routine (Giemsa) stained sections of gastric mucosa obtained at endoscopy.
2) **Rapid urease test:** Cheap, quick, specific (>95%) with sensitivity of 85%. Can produce false negative if pt is on PPI. Gastric biopsies, usually antral unless additional material is needed to exclude proximal migration, are added to a substrate containing **urea and phenol red** . If H. pylori is **present**, the urease enzyme that the bacteria produce splits the urea to release ammonia, which raises the pH of the solution and causes a rapid colour change (**yellow to red**).
3) **Microbiological culture: GOLD standard,** This technique is typically used for patients with refractory H. pylori infection to identify the appropriate antibiotic regimen; routine culture is rare.
4) **PCR**

◀ Indications for H. Pylori eradication:

- **All** patients with proven ulcers who are **H. pylori-positive should be offered eradication** as primary therapy.
- **When is surgery indicated in PUD?**
  - In cases of emergency (**Perforation** or **Hemorrhage**)
  - Or elective (**Gastric flow obstruction**, **Persistent ulceration despite adequate medical therapy** or **Recurrent ulcer following gastric surgery**)

i 21.36 Indications for *Helicobacter pylori* eradication

Definite

- Peptic ulcer
- Extranodal marginal-zone lymphomas of MALT type
- Family history of gastric cancer
- Previous resection for gastric cancer
- *H. pylori*-positive dyspepsia
- Long-term NSAID or low-dose aspirin users
- Chronic (>1 year) PPI users
- Extragastric disorders:
  - Unexplained vitamin B_{12} deficiency*
  - Idiopathic thrombocytopenic purpura*
  - Iron deficiency anaemia* (see text)

Not indicated

- Gastro-oesophageal reflux disease
- Asymptomatic people without gastric cancer risk factors

*If *H. pylori*-positive on testing.
(MALT = mucosa-associated lymphoid tissue; NSAID = non-steroidal anti-inflammatory drug; PPI = proton pump inhibitor)

Management of PUD

◀ European Helicobacter Pylori study group guidelines

- **Triple therapy** with **omeprazole** (20 mg twice daily), **amoxicillin** (1 g twice daily), and **clarithromycin** (500 mg twice daily) **for 7 to 14 days.**
- A longer duration of treatment (14 versus 7 days) may be more effective in curing infection but this remains controversial. more than 14 days is not recommended

◀ Treatment regimens

Regimen[1]	comment
• (Triple therapy) **PPI**, **amoxicillin** 1 gm, **clarithromycin** 500 mg all twice daily for 7-14 days.	• 1st line treatment [2] regimen of choice (can substitute metronidazole 500 mg twice daily for amoxicillin but only in penicillin allergic patients) metronidazole has bad taste.
• (Quadruple therapy) **Bismuth** 525 mg, **metronidazole** 500 mg, **tetracycline** 500 mg all four times daily with **PPI** twice daily for 7-14 days. Please Make Tummy Better	• Can be used as 1st line treatment (7-14 days) but generally reserved for retreatment (14 days) Quadruple therapy can be used when there is resistance to clarithromycin
• PPI, amoxicillin 1 gm, metronidazole 500 mg all twice daily for 14 days	• 1st line treatment in macrolide allergic patients and retreatment if failed 1st line treatment of choice

Treatment Regimen	Duration (days)	Eradication Rate (%)
Omeprazole 20 mg BID + **Amoxicillin** 1 g BID + **Clarithromycin** 500 mg BID	14	80 - 86
Lansoprazole 30 mg BID + **Amoxicillin** 1 g BID + **Clarithromycin** 500 mg BID	10 - 14	86
Bismuth subsalicylate 525 mg QID + **Metronidazole** 250 mg QID + **Tetracycline** 500 mg + **PPI**	PPI for another 14 taken OD or BID)	80

OD = Once a day. | **BID** = twice a day. | **QID** = 4 times a day.

1- all of them are 1st line therapy. But it depends on the region.

2- According to Dr this is **NOT** the first line therapy in saudis because of high rates (>20%) of clarithromycin resistance!
It is the first line of treatment if there is no clarithromycin resistance.

SO Bottom line, what is the first line in our region? (Quadruple therapy) **Bismuth**, **metronidazole**, **tetracycline**, **PPI for 14 days**

Management PUD *cont'*

◀ Clarithromycin-resistant bacteria

- Pooled data from 20 studies involving 1975 patients treated with standard triple therapy showed an eradication rate of 88% in clarithromycin-sensitive strains vs 18% in clarithromycin-resistant strains.
- **A 10-day sequential regimen**
  - **First 5 days: PPI** and **amoxicillin** 1 g, each given twice daily.
  - **second 5 days: PPI, clarithromycin 500 mg,** and **tinidazole** 500 mg, each given twice daily.
- Improved overall eradication rates compared with standard PPI triple therapy (89% vs. 77 %), but was particularly better for clarithromycin-resistant bacteria (**89%** vs. 29%).

◀ Concomitant therapy[1]

- Novel regimen which was proved successful in the presence of clarithromycin resistance. This is a **4-drug regimen** containing a **PPI** ,**clarithromycin** (500 mg, b.i.d.), **amoxicillin** (1 g, b.i.d.) and **metronidazole** (500 mg, b.i.d.) which are all given for the entire duration of therapy.

		Eradicated		
Analysis Population	N	N	Percent	95% CI for Percent Eradicated
Intention to Treat (ITT)	1463			
14-day Standard	488	401	82.2%	78.5%, 85.5%
5-day Concomitant	489	360	73.6%	69.5%, 77.5%
10-day Sequential	486	372	76.5%	72.5%, 80.2%

◀ Rescue therapy[2]:

Regimen	comment
• **PPI**, **levofloxacin** 250 to 500 mg, **amoxicillin** 1 gm all twice daily **for 14 days**	• "Rescue" therapy for those failing **two course** of above treatments
• **PPI**, **rifabutin** 150 mg, **amoxicillin** 1 gm all twice daily for 14 days	• Alternative "rescue" therapy
• Based on culture	• If all medications listed above didn't work then we do culture. Why usually we don't do culture? because it takes time, we usually do UBT, endoscopy and routine histopathology

- Poor compliance with medication, and patient demographics such as younger age, smoking, prior antibiotic use, and underlying condition (functional dyspepsia vs. peptic ulcer).
- Some patients don't continue their therapy course, why?
  - They think they are already cured (but actually it was PPI relief)
  - They want to stop because of therapy's side effects

1- first line along with quadruple therapy in Saudi Arabia (if both are choices in the exam, choose quadruple)

2- When some patients had resistance for sequential therapy, they came up with rescue therapy.

Peptic Ulcer Disease (PUD) *cont'*

◀ Complications of PUD:

	Clinical findings	Diagnostic studies	Management	Other
Perforation	**Acute, severe abdominal pain**, signs of peritonitis, hemodynamic instability	**Upright CXR (free air under diaphragm),** CT scan is the most sensitive for perforation (detects free abdominal air)	**Emergency surgery** to close perforation and perform definitive ulcer operation (such as highly selective vagotomy or truncal vagotomy/pyloroplasty)	Can progress to sepsis and death if untreated
Gastric outlet obstruction	**Nausea/vomiting** (poorly digested food), epigastric fullness/early satiety. weight loss	**Barium swallow and upper endoscopy**; saline load test (empty stomach with as nasogastric tube, add 750mL saline, aspirate after 30min - test is positive if aspirate >400mL	**Initially, nasogastric suction**; replace electrolyte/ volume deficits; supplement nutrition if obstruction is long standing. **Surgery is eventually necessary in 75% of patients**	Most common with duodenal ulcers and type III gastric ulcers
GI bleeding	Bleeding may be slow (leading to **anemic symptoms**) or can be rapid and severe (leading to shock)	Stool guaiac, **upper GI endoscopy** (diagnostic and therapeutic)	**Resuscitation**; diagnose site of bleed via endoscopy and treat; **perform surgery for acute bleeds** that require transfusion of 6U or more of blood	**PUD is the most common cause of upper GI bleeding**

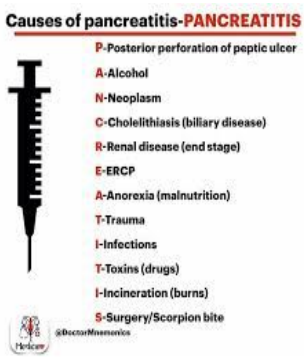
Acute pancreatitis

Introduction

- There is inflammation of the pancreas resulting from prematurely activated pancreatic digestive enzymes that invoke pancreatic tissue autodigestion.
- Most patients with acute pancreatitis have mild to moderate disease but up to 25% have severe disease.
- There are two forms of acute pancreatitis, mild and severe:
  a. **Mild acute pancreatitis** is most common and responds well to supportive treatment.
  b. **Severe acute pancreatitis** (necrotizing pancreatitis) has significant morbidity and mortality.

◀ Causes

- **Gallstones (40%)**—The gallstone passes into the bile duct and blocks the ampulla of Vater. "ALWAYS consider gallstone pancreatitis and rule it out even in pt with hx of alcohol use"
- **Alcohol abuse (40%)**
- Post-ERCP[1] —Pancreatitis occurs in up to 10% of patients undergoing ERCP[2] .
- Viral infections (e.g., mumps, Coxsackievirus B)
- Drugs: Sulfonamides, thiazide **diuretics,NSAIDS,** furosemide, estrogens, HIV medications.
- Postoperative complications. (high mortality rate)
- Scorpion bites.
- Pancreas divisum[3].
- Pancreatic cancer.
- Hypertriglyceridemia, hypercalcemia.
- Uremia.
- Blunt abdominal trauma **(most common cause of pancreatitis in children)**



★ Clinical Features

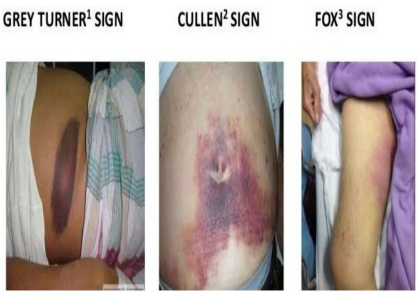
Symptoms:

1. Abrupt onset of severe abdominal pain, usually in the epigastric region.
- **May radiate to back** (50% of patients).
- Often steady, dull, tenderness and severe; worse when supine and after meals

2. Nausea and **vomiting**, anorexia

Signs:

1. Low-grade fever, tachycardia, hypotension, leukocytosis.
2. Epigastric tenderness, abdominal distention.
3. Decreased or absent bowel sounds indicate partial ileus.
4. The following signs are seen with hemorrhagic-pancreatitis as blood tracks along fascial planes:
   - ➢ **Grey Turner's sign** (flank ecchymoses)
   - ➢ **Cullen's sign** (periumbilical ecchymoses)
   - ➢ **Fox's sign** (ecchymosis of inguinal ligament)



Signs of severe Necrotizing Pancreatitis:
- Cullen sign: Blue discoloration around the umbilicus -> due to hemoperitoneum.
- Turner's sign: Bluish purple discoloration of flanks -> tissue catabolism of Hb

1- "**Endoscopic retrograde cholangiopancreatography**"
2- Presumably because of back pressure from injection of contrast material into the ductal system. Most people have asymptomatic increase in amylase, only 2-8% of pt will actually develop symptomatic pancreatitis.
3- Is a congenital anomaly in the anatomy of the ducts of the pancreas in which a single pancreatic duct is not formed, but rather remains as two distinct dorsal and ventral ducts.

Acute pancreatitis

◄ Diagnosis

1 Laboratory studies:

- **Serum amylase** is the most common test **(Best initial test)**, but many conditions cause hyperamylasemia (nonspecific) and its absence does not rule out acute pancreatitis (nonsensitive). However, if levels are more than five times the upper limit of normal, there is a high specificity for acute pancreatitis.
- **Serum lipase**—**(more specific for pancreatitis than amylase).**
- LFTs. "To identify cause (gallstone pancreatitis)."
- Hyperglycemia, hypoxemia, and leukocytosis may also be present.
- Order the following for assessment of prognosis **(Ranson's criteria):** glucose, calcium, hematocrit, BUN, arterial blood gas (PaO2, base deficit), LDH, AST, WBC count

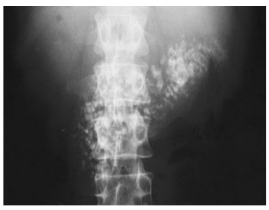
TABLE 3-3 Ranson's Criteria

Admission Criteria (GA LAW)	Initial 48 Hours Criteria (C HOBBS)	Mortality
Glucose >200 mg/dL	Calcium <8 mg/dL Decrease in Hematocrit >10%	<3 criteria—1%
Age >55 years	Pa_{O_2} <60 mm Hg	3–4 criteria—15%
LDH >350	BUN increase >8 mg/dL	5–6 criteria—40%
AST >250	Base deficit >4 mg/dL	>7 criteria—100%
WBC >16,000	Fluid sequestration >6 L	

2 Radiological studies:

1. Abdominal radiograph:

- Has a limited role in the diagnosis of acute pancreatitis.
- More helpful in **ruling out** other diagnoses, such as intestinal perforation (free air).
- The presence of calcifications can suggest chronic pancreatitis.
- if severe; "**sentinel loop**" or "**colon cutoff sign**" may be seen.



2. Abdominal ultrasound:

- Can help in **identifying cause** of pancreatitis (e.g., gallstones).
- Useful for following up pseudocysts or abscesses.

3. CT scan of the abdomen:

- **Most accurate test** for diagnosis of acute pancreatitis and for identifying complications of the disease."
- Indicated in patients with severe acute pancreatitis.

4. ERCP (indications):

- Severe gallstone pancreatitis with **biliary obstruction**.
- To identify uncommon causes of acute pancreatitis **if disease is recurrent**.

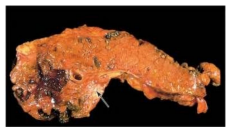
Acute pancreatitis

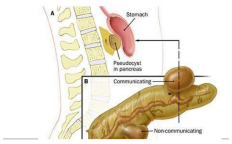
Complications

1. Pancreatic necrosis (may be sterile or infected):

- **Sterile pancreatic necrosis**—Half of all cases resolve
- spontaneously. Should be monitored closely in an ICU.
- **Infected pancreatic necrosis**—has high mortality rate (results inmultiple organ failure in 50% of cases); surgical débridement and antibiotics indicated.

The only way to distinguish sterile from infected necrosis is via CT-guided percutaneous aspiration with Gram stain/culture of the aspirate.



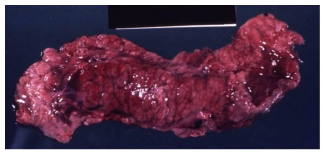


2. Pancreatic pseudocyst:

- Encapsulated fluid collection that appears 2 to 3 weeks after an acute attack— unlike a true cyst, it lacks an epithelial lining.
- Complications of untreated pseudocysts include rupture, infection, gastric outlet obstruction, fistula, hemorrhage into cyst, and pancreatic ascites.
- Diagnosis: CT scan is the study of choice.
- Treatment:
  - ➢ Cysts <5 cm: observation.
  - ➢ Cysts >5 cm: drain either percutaneously or surgically.

3. Hemorrhagic pancreatitis:

- Characterized by Cullen's sign, Grey Turner's sign, and Fox's sign.
- CT scan with IV contrast is the study of choice.



4. Adult respiratory distress syndrome —a life-threatening complication with high mortality rate.

5. Pancreatic ascites/pleural effusion. "The most common cause is inflammation of peritoneal surfaces."

6. Ascending cholangitis. "Due to gallstone in ampulla of Vater, leading to infection of biliary tract"

7. Pancreatic abscess (rare). "Develops over 4 to 6 weeks and is less life threatening than infected pancreatic necrosis."

Treatment

1. Patients with mild acute pancreatitis:

- Bowel rest (NPO).
- IV fluids. "Correct electrolyte abnormalities."
- Pain control.
- Nasogastric tube. "If severe nausea/vomiting or ileus present."

2. Patients with severe pancreatitis:

- Should be admitted to the ICU.
- Early enteral nutrition in the first 72 hours is recommended through a nasojejunal tube.
- If the severe acute pancreatitis has not resolved in a few days, supplemental parenteral nutrition should be started.
- If more than 30% of the pancreas is necrosed, prophylactic antibiotics (imipenem) should be considered to prevent infection. "Which has high morbidity and mortality"

Irritable bowel syndrome (IBS)

What is IBS?

Definition

★ Irritable bowel syndrome (IBS) is an intestinal disorder that causes abdominal pain or discomfort, cramping or bloating, and diarrhea or constipation. Irritable bowel syndrome is a long-term but manageable condition.

- It is predominantly a pain syndrome of unknown etiology that is often relieved by bowel movement.
- Educate patient that this is a chronic disease that stays for life.

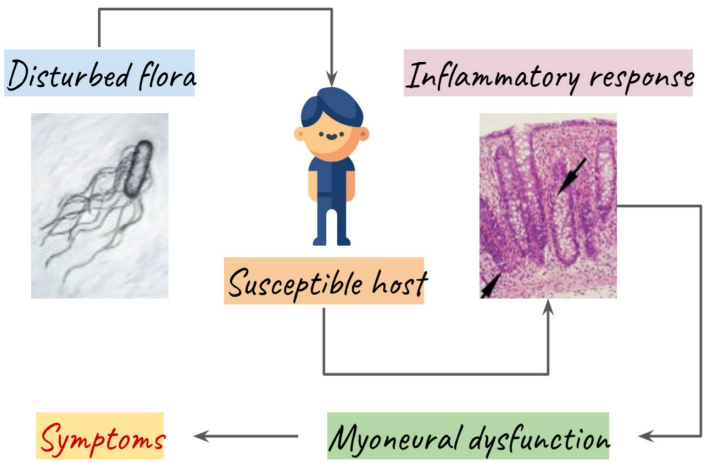
Who gets IBS?

- It is estimated that between 10% and 15% of the population of North America, or approximately 45 million people, have irritable bowel syndrome.
- Only about **30%** of them will consult a doctor about their symptoms.
- IBS tends to be more common in In women, IBS is 2 to 3 times more common than in men.
- Young female women, Type A personality
- IBS is sometimes associated with a **history of physical or sexual abuse** and this is an important aspect of the history as these patients benefit from psychologically based therapy.

IBS pathophysiology

There is no clear pathophysiology but these are hypotheses:

- **Heredity;** nature vs nurture
- Dysmotility, "spasm"
- Visceral Hypersensitivity (they feel every bowel movement)
- Altered CNS perception of visceral events
- Psychopathology
- Infection/Inflammation
- Altered Gut Flora



Mucosal Compartment

- Frank inflammation
- Immune Activation
  - ↑ IEL's
  - ↑ CD3+, CD25+
- Decreased IgA + B Cells
- Altered expression of genes involved in mucosal immunity

IBS Subtypes ★

Diarrhoea predominant.	**Constipation** predominant.	**Pain** predominant.

- Most patients **alternate between** episodes of **diarrhoea**, **constipation** and pain. but it is useful to classify them as having predominantly constipation or predominantly diarrhoea.
- Those with **constipation** tend to pass **infrequent (less than 3) pellety stools**, usually in association with abdominal pain or proctalgia. Those with **diarrhoea** have **frequent defecation (more than 3)** but produce **low-volume** stools and rarely have nocturnal symptoms.

Irritable bowel syndrome (IBS) *cont'*

◀ Clinical features

- The most common presentation is that of **recurrent abdominal discomfort**. This is usually **colicky** or **cramping in nature**, felt in the lower abdomen and **relieved by defecation.**
- Patients **do not lose weight** and are constitutionally well.
- Physical examination is generally **unremarkable**, with the exception of variable tenderness to palpation.
- **Associated symptoms:**
  - In people with IBS in hospital OPD.
    - 25% have **depression**, 25% have **anxiety**.
  - In one study 30% of women IBS sufferers have **fibromyalgia**

◀ Rome III diagnostic criteria:

❖ At least **12 weeks** history, which needs not be consecutive in the last **12 months** of abdominal discomfort or pain that **has 2 or more of the following:**

Relieved by defecation.	**Onset associated with change in stool frequency.**	**Onset associated with change in form (appearance) of stool.**

General:

- Feeling of incomplete evacuation.
- Abdominal fullness , bloating or swelling [1]
- Passage of mucus per rectum. but rectal bleeding does not occur

◀ Alarm features in IBS

1. **Age >50 years; male gender**
2. **Weight loss**
3. **Nocturnal symptoms**
4. **Family history of colon cancer**
5. **Anemia**
6. **Rectal bleeding**

1- Abdominal bloating worsens throughout the day; the cause is unknown but **it is not** due to excessive intestinal gas.

Irritable bowel syndrome (IBS)

◀ Differential diagnosis:

IBD	Cancer	Diverticulosis	Endometriosis	Celiac disease

◀ Assessment:

- History: type of pain, location, associated symptoms, constitutional symptoms
- **Results should be normal or non-specific.**
- Abdomen and rectal examination.

◀ Diagnostic testing in IBS

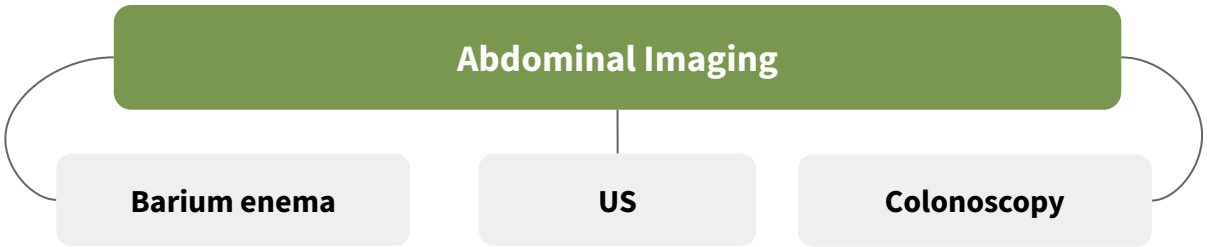
1- CBC, LFT	2- Stool analysis	3- Thyroid function test[1]	4- Celiac Ab

Note: **Current best evidence does not support the routine use of blood tests to exclude organic gastrointestinal disease in patients who present with typical IBS symptoms without alarm features.**

◀ Hydrogen Breath Testing:

- **Lactose malabsorption (typically diagnosed via abnormal hydrogen breath testing)** is estimated to be approximately 25% in western countries and perhaps as high as 75% worldwide. it is underdiagnosed condition, make sure you ask about it in your history. If you suspect it do not forget to request hydrogen breath test

◀ Abdominal Imaging:



- **Prevalence of colorectal cancer in these studies was <u>low</u>, ranging from 0 to 0.51%.**
- Abdominal imaging is usually not used in IBS patients, If you take good history, good physical, basic workup and all of them are normal in a susceptible pt, no need to do these expensive procedures

1. Should be tested in most of GI symptoms

Referral & Management of IBS

◀ Reasons to Refer

Reasons to Refer	Urgent Referral
• Age > 45 years at onset. • Family history of bowel cancer. • Failure of primary care management. • Uncertainty of diagnosis. • Abnormality on examination or investigation	• Constant abdominal pain • Constant diarrhea • Constant distension • Rectal bleeding • Weight loss or malaise.

◀ Management of IBS:

➔ **Patients' concerns (The most important thing is patient - doctor relationship) ~50% of the treatment**
➔ **Explanation**
➔ **Treatment approaches**
- ◆ Placebo effect of up to 70% in all IBS treatments.
- ◆ Treatment should depend on symptom sub-type.

◀ Antidepressants:

- Poor evidence for efficacy.
- Better evidence for tricyclics. (if it is pain predominant you might give little TCA)
- Very little evidence for SSRIs

SSRIs

- Six studies have been conducted to date, two each involving fluoxetine, paroxetine and citalopram.
- Most patients noted an improvement in overall wellbeing, although none of the studies showed any benefit with regards to bowel habits, and abdominal pain was generally not improved.

◀ If IBS is Pain Predominant:

➔ **Antispasmodics** will help 66%.
➔ **Mebeverine** is probably first choice.
➔ **Hyoscine** 10mg qid can be added.

- A recent meta-analysis of 22 studies involving 1778 patients and 12 different antispasmodic agents demonstrated modest improvements in global IBS symptoms and abdominal pain
- However, up to 68% of patients suffered side effects when given the high dose required to improve abdominal pain

Management of IBS

◄ Constipation and Diarrhea Predominant IBS

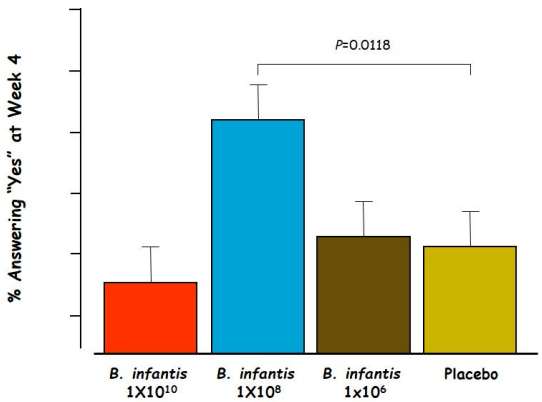
Constipation Predominant IBS:	Diarrhea Predominant IBS:

• **Lifestyle Modifications** (exercise and diet)
• A lot of water
• **Bowel Training and Education**
• **Fibre**[1]
  ○ Twelve randomized controlled trials have been performed to date evaluating the efficacy of fiber in the treatment of IBS. Four of these studies noted an improvement in stool frequency (polycarbophil and ispaghula husk), while one noted an improvement in stool evacuation
  ○ No improvement in abdominal pain
  ○ 30-50% of patients treated with a fiber product will have a significant increase in gas
• **Lubiprostone** stimulates type 2 chloride channels in epithelial cells of the gastrointestinal tract thereby causing an efflux of chloride into the intestinal lumen
Why chloride channels? when chloride is not absorbed thus sodium also is not absorbed, when sodium is not absorbed water is not absorbed thus this may relief constipation
  ○ It was approved by the FDA for the treatment of adult men and women with chronic constipation in January 2006
  ○ Nausea and diarrhea 6-8%
	• **Increasing dietary fibre is sensible advice.**
	• **Fibre varies, 55% of patients will get worse with bran.**
	• **Loperamide** inhibiting intestinal secretion and peristalsis, loperamide slows intestinal transit and allows for increased fluid reabsorption, thus improving symptoms of diarrhea
	• **Alosetron** is 5-HT3 receptor antagonist that slows colonic transit
	  ○ Meta-analysis of eight randomized controlled trials involving 4842 patients determined that **alosetron** provided a significant reduction in the global symptoms of diarrhea, abdominal pain, and bloating in patients with IBS and diarrhea
	  ○ Four-fold increased risk of ischemic colitis compared

◄ Probiotics

- **Mode of Action of Probiotics?**
  - Competition with, and exclusion, of pathogens
  - Anti-bacterial:
    - Produce bacteriocins
    - Destroy toxins
  - Enhance barrier function, motility
  - Enhance host immunity by :
    - Immune modulation
    - Cytokine modulation
    - IgA production
  - Metabolic functions

❖ **Global assessment of symptom relief:**



1- Actually fibers are used in both diarrhea and constipation… what is the difference? It depends on the amount of water you take with the fiber. If you take too much water with fiber then it works as a lubricant and improves bowel movement. While if you take fibers with minimal amount of water (as adding fibers to yoghurt then fibers work as a sponge and make stool bulky

Management of IBS

◀ What about diet?

- Avoid caffeine.
- Limit your intake of fatty foods.
- If diarrhea is your main symptom, limit dairy products, fruit, or the artificial sweetener sorbitol.
- Increasing fiber in your diet may help relieve constipation.
- Avoiding foods such as beans, cabbage, or uncooked cauliflower or broccoli can help relieve bloating or gas.

i 21.65 Dietary management of irritable bowel syndrome

- Eat regularly and avoid missing meals
- Take time to eat
- Ensure adequate hydration and avoid carbonated and caffeinated drinks
- Reduce alcohol intake
- Reduce intake of 'resistant' starch and insoluble fibre
- Avoid foods with artificial sweeteners
- Consider a wheat-free diet
- Consider a lactose exclusion diet
- Consider a diet low in FODMAPs

(FODMAPs = fermentable oligo-, di- and monosaccharides, and polyols)

◀ Alternative and complementary medicine:

- Peppermint, germanium, lavender oils
- RCT of 57 IBS patients randomized to receive either peppermint capsules or placebo demonstrated a significant benefit for the peppermint-treated group after 4 weeks.
- Seventy-five percent of the study group versus 38% of the placebo group reported a greater than **50%** reduction in total IBS symptoms

◀ Alternative Medicine:

- **Hypnosis:** Hypnosis can help some people relax, which may relieve abdominal pain.
- **Relaxation or meditation:** Relaxation training and meditation may be helpful in reducing generalized muscle tension and abdominal pain.
- **Biofeedback:** Biofeedback training may help relieve pain from intestinal spasms. It also may help improve bowel movement control in people who have severe diarrhea.

❖ Self-help: **IBS network - IBS support group - Awareness**

i 21.66 Complementary and alternative therapies for irritable bowel syndrome

Manipulative and body-based	
• Massage, chiropractic	
Mind–body interventions	
• Meditation, hypnosis*, cognitive therapy	
Biologically based	
• Herbal products*, dietary additives, probiotics*	
Energy healing	
• Biofield therapies (reiki), bio-electromagnetic field therapies	
Alternative medical systems	
• Ayurveda, homeopathy, traditional Chinese medicine	

*Some evidence for benefit exists.
From Hussain Z, Quigley EMM. Systematic review: complementary and alternative medicine in the irritable bowel syndrome. Aliment Pharmacol Ther 2006; 23:465–471.

◀ Algorithm for Management of IBS:

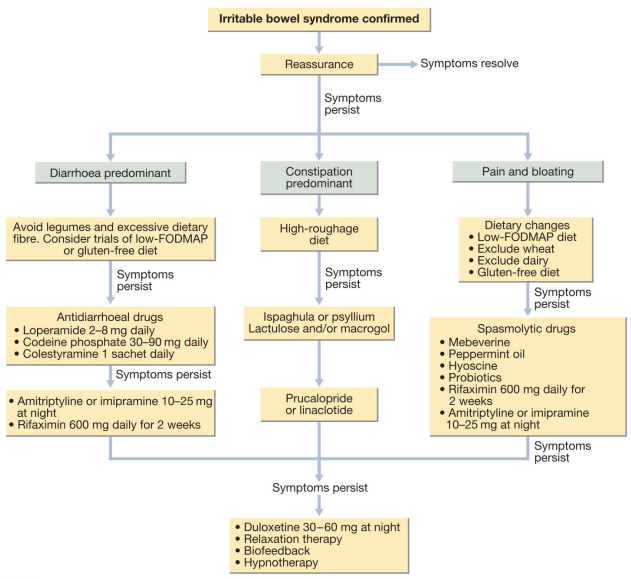


Fig. 21.54 Management of irritable bowel syndrome. (FODMAP = fermentable oligo-, di- and monosaccharides, and polyols)

◄ Case study 1

Q1: A 34 y/o lady who comes to your clinic because of epigastric pain since 5 months ago. She complains of bloating and early satiety too. There is no alarm symptom in her history. She use no medications. Her physical examination is normal.

- What is the most important information from this scenario?

- What is the most likely diagnosis?

A- Dyspepsia
B- Peptic ulcer disease
C- Pancreatitis
D- Gastric cancer

- What is the best next step?

A- Endoscopy
B- High dose PPI
C- H pylori testing
D- Ultrasound abdomen

What is the most important information from this scenario?

1- Age
2- Duration, it's chronic pain
3- No alarming symptoms

What is the most likely diagnosis? Answer: A

What is the best next step?

Answer: C

A- Endoscopy (no alarming symptoms in this case so not the best answer)
B- High dose PPI (If we are in an area with low prevalence of H pylori, we ho with PPI)
C- H pylori testing (the best answer according to this case, and in our culture H.pylori is prevalent so it is better to do it first)
D- Ultrasound abdomen (not the first line of investigation)

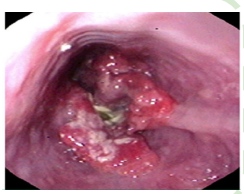
- **So, we start with H pylori testing, if the test is -ve we give high dose PPI. if it didn't work we go for endoscopy**

◄ Case study 2

A patient was a 60 y/o lady who was referred to me because of constant epigastric pain. She mentioned 6 kg wt loss since 3m ago. She was anemic with ferritin =5. What is the best diagnostic test?

A- Endoscopy
B- High dose PPI
C- H pylori testing
D- Ultrasound abdomen

Answer: A, because of the presence of alarming symptoms in this case as weight loss and anemia. **When do we do endoscopy?** In presence of alarming symptoms or when not responding to treatment or age >55.



◀ Case study 3

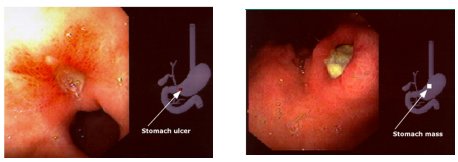
A 44 y/o lady who was referred to me because of chronic epigastric pain mainly at night and vomiting. She gave a history of one day history of melena but no other alarm symptoms. What is the next step?

A- Endoscopy
B- High dose PPI
C- H pylori testing
D- Ultrasound abdomen

Answer: A,

A 44 y/o (middle age**) lady who was referred to me because of chronic epigastric pain mainly at night** (you need to think about **structural or organic** disease rather than functional) **and vomiting. She gave a history of one day history of melena** (indicate upper GI bleeding**) but no other alarm symptoms. What is the next step?**

A- Endoscopy
B- High dose PPI
C- H pylori testing
D- Ultrasound abdomen



◀ Case study 4

A 30 years old lady with chronic abdominal pain mainly central associated with bloating . Alternating bowel habit and history of passing mucus with loose motions no Wt loss no blood/rectum. What is the likely diagnosis?

A- Pancreatitis
B- PUD
C- IBS
D- Gastric cancer

Answer: C,

- **We get from the history : Age (not old), chronicity, central with Alternating bowel habit and no alarming symptoms.**

You did it <3

Summary

Dyspepsia	
Clinical Approach:	
History	• Ulcer-like or acid dyspepsia • Dysmotility-like dyspepsia • Unspecified dyspepsia
Physical examination	• usually normal
Routine laboratory tests:	
Routine blood count	
Blood chemistry	
Indications for endoscopy	
Age >55	
Presence of alarming symptoms	• Unintended weight loss • Persistent vomiting • Progressive dysphagia • Odynophagia • Hematemesis • **Unexplained anemia or iron deficiency** • Palpable abdominal mass or lymphadenopathy • Family history of upper gastrointestinal cancer • Previous gastric surgery • Jaundice
Irritable bowel syndrome	
Clinical Feature	• Recurrent Cramping abdominal pain characterized by "Relieved by defecation and Less at night" • Change frequency and consistency of stool • Abnormal stool passage Bloating
Diagnosis "clinical diagnosis of exclusion"	• Rome III criteria • Manning's Criteria. • Rome II Diagnostic Criteria • Ask about Alarm symptoms that suggest other serious diseases to exclude them
Reasons to Refer	• Age > 45 years at onset. • Family history of bowel cancer. • Failure of primary care management. • Uncertainty of diagnosis. • Abnormality on examination or investigation.
Urgent Referral	• Constant abdominal pain. • Constant diarrhoea. • Constant distension. • Rectal bleeding. • Weight loss or malaise.

Summary

Management of H.pylori	
Clarithromycin-resistant	
10-day sequential regimen	• "**PPI**+**amoxicillin**" 2/day for the first 5 days, followed by "**PPI**+ **clarithromycin**+ **tinidazole**" each given 2/day for the remaining 5 days
Clarithromycin-sensitive	
1st line treatment regimen of choice	• "**PPI**, **amoxicillin**, **clarithromycin**" twice daily for 7-14 days
Penicillin allergy	• "**PPI**, **metronidazole**, **clarithromycin**" twice daily for 7-14 days
Macrolide allergy	• "**PPI**, **amoxicillin**, **metronidazole**" twice daily for 14 days
generally reserved for retreatment	• "**Bismuth**, **metronidazole**, **tetracycline**" 4 times daily + PPI twice daily for 7-14 days
H.pylori as a cause of PUD	
Diagnosis	
Non endoscopic methods	• Serum antigen • Urea breath test • Stool antigen
Endoscopic methods	• Histology • Rapid urease test • Culture • PCR

Lecture Quiz

1. You see a 47-year-old man in clinic with a three-month history of epigastric dull abdominal pain. He states that the pain is worse in the mornings and is relieved after meals. On direct questioning, there is no history of weight loss and the patient's bowel habits are normal. On examination, his abdomen is soft and experiences moderate discomfort on palpation of the epigastric region. The most likely diagnosis is:

A. Gastric ulcer
B. Gastro-oesophageal reflux disease (GORD)
C. Duodenal ulcer
D. Gastric carcinoma
E. Gastritis

2. You see an 80-year-old man who presents to accident and emergency with epigastric pain. The pain started 3 days ago and today he noticed that the colour of his stools has changed to a 'tarry-black' colour. Associated symptoms include nausea and lethargy. The patient is a smoker of 20 cigarettes a day and has recently finished eradication treatment for a duodenal ulcer. The patient is alert and orientated with a pulse rate of 99 and blood pressure of 98/69, respiratory rate of 18, oxygen saturations of 98% on room air and temperature of 37.2°C. On examination, the abdomen is soft with marked tenderness in the epigastric region and bowel sounds are present. The rectum is empty, on PR examination, with some traces of malaena. The patient has been started on high flow oxygen and has been given some oral analgesia. The most appropriate next step in managing this patient is:

A. Keep nil by mouth and arrange endoscopy
B. Request an erect chest x-ray
C. Intravenous pantoprazole
D. ECG
E. Intravenous cannulation and fluids

3. A 32-year-old white woman complains of abdominal pain off and on since the age of 17. She notices abdominal bloating relieved by defecation as well as alternating diarrhea and constipation. She has no weight loss, GI bleeding, or nocturnal diarrhea. On examination, she has slight LLQ tenderness and gaseous abdominal distension. Laboratory studies, including CBC, are normal. Which of the following is the most appropriate initial approach?

A. Recommend increased dietary fiber, antispasmodics as needed, and follow-up examination in 2 months.
B. Refer to gastroenterologist for colonoscopy.
C. Obtain anti-endomysial antibodies.
D. Order UGI series with small bowel follow-through.
E. Order small bowel biopsy.

Q4: A 35-year-old woman has chronic crampy abdominal pain and intermittent constipation and diarrhea, but no weight loss or gastrointestinal bleeding. Her abdominal pain is usually relieved with defecation. Colonoscopy and upper endoscopy with biopsies are normal, and stool cultures are negative. Which of the following is the most likely diagnosis?
A- Infectious colitis
B- Irritable bowel syndrome
C- Crohn disease
D- Ulcerative colitis

Q5: A 42-year-old overweight but otherwise healthy woman presents with sudden onset of right-upper abdominal colicky pain 45 minutes after a meal of fried chicken. The pain is associated with nausea and vomiting, and any attempt to eat since has caused increased pain. Which of the following is the most likely cause?
A. Gastric ulcer
B. Cholelithiasis
C. Duodenal ulcer
D. Acute hepatitis

Q6: Which of the following is the most accurate statement regarding H pylori infection?
A. It is more common in developed than underdeveloped countries.
B. It is associated with the development of colon cancer.
C. It is believed to be the cause of nonulcer dyspepsia.
D. The route of transmission is believed to be sexually transmitted.
E. It is believed to be a common cause of both duodenal and gastric ulcers.

Q7: A 45-year-old man was brought to the ER after vomiting bright red blood. He has a blood pressure of 88/46 mm Hg and heart rate of 120 bpm. Which of the following is the best next step?
A. Intravenous fluid resuscitation and preparation for a transfusion
B. Administration of a proton-pump inhibitor
C. Guaiac test of the stool
D. Treatment for H pylori

Q8: Which one of the following patients should be promptly referred for endoscopy?
A. A 65-year-old man with new onset of epigastric pain and weight loss
B. A 32-year-old patient whose symptoms are not relieved with ranitidine
C. A 29-year-old H pylori-positive patient with dyspeptic symptoms
D. A 49-year-old woman with intermittent right-upper quadrant pain following meals

Answers: Q1:C | Q2:E | Q3:A | Q4:B | Q5:B | Q6:E | Q7:A | Q8:A

Answers Explanation File!

GOOD LUCK !

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