Abdominal Pain Including IBS

Objectives:

- Causes of Abdominal pain
- Functional dyspepsia
- Approach to management of dyspepsia
- Management of H pylori
- Irritable bowel syndrome

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- Editing file
- Feedback

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 cause → More chronic usually it is chronic, sometimes patients think pain just started but it was chronic

- 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"

Dyspepsia

Dyspepsia: is a collection of symptoms including nausea bloating and epigastric pain
Functional Dyspepsia: we mean when there is no pathology (no problem in serology, no problem in radiology) but it is not functioning well.

Epidemiological data about dyspepsia:

- Estimated Prevalence of dyspepsia is between 20-40%.
- Most affected people do not seek medical care.
- 25 and 40% of Individuals with dyspepsia will consult a PCPS.

Definition: (Rome III Committee)

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

- Postprandial distress syndrome (PDS) (feeling nausea, bloating or early satiety after a meal)
- Epigastric pain syndrome (EPS) which indicate meal-related and unrelated symptoms. (epigastric pain either they have eaten or not)

Etiology:

Diagnosis
Functional dyspepsia (up to 60 percent)
Dyspepsia caused by structural or biochemical disease
Peptic ulcer disease
Gastroesophageal reflux disease (GERD)
Biliary pain
Chronic abdominal wall pain
Gastric or esophageal cancer
Gastroparesis
Pancreatitis
Carbohydrate malabsorption
Medications (including potassium supplements, digitalis, iron, theophylline, oral antibiotics [especially ampillin and erythromycin], NSAIDs, corticosteroids, niacin, gemfibrozil, narcotics, colchicine, quinidine, estrogens, levodopa)
Infiltrative diseases of the stomach (eg, Crohn's disease sarcoidosis)
Metabolic disturbances (hypercalcemia, hyperkalemia)
Hepatoma
Ischemic bowel disease
Systemic disorders (diabetes mellitus, thyroid and parathyroid disorders, connective tissue disease)
Intestinal parasites (Giardia, Strongyloides)
Abdominal cancer, especially pancreatic cancer

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

Clinical Approach:

History:

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

Note that: *Peptic ulcer* pain is worsened by food (because food stimulates acid secretion) while *duodenal ulcer* pain is relieved by food

Physical examination: usually normal

- A palpable mass.

Alarm Symptoms:

- Unintended weight loss or fever, chills, night sweats
- 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
- 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%)

Routine laboratory tests:

- Routine blood count (check if anemic)
- Blood chemistry
- Invasive procedure Endoscopy:
    - Gold standard test to exclude (organic causes) gastroduodenal ulcers, reflux esophagitis, and upper gastrointestinal cancers.
    - Beneficial because up to 40 percent of patients have an organic cause of dyspepsia.
    - It also provides reassurance to patients يبعد عن المرضى الوسوسة بأن فيهم شي
    - Disadvantages:
        - Expensive.
        - Invasive.
        - Not cost-effective in young patients without alarm symptoms.
        - Up to 50% are normal (so not cost effective)

Case 1:

- Patient is a 34 y/o lady who comes to your clinic because of epigastric pain since 5m ago
- She complains of bloating and mainly early satiety too
- There is no alarm symptom in her history, She use no drug
- Her physical examination is normal

What is the most important information from this scenario ? 1- Age 2-Duration , it's chronic pain 3- No alarming symptoms

What do you do for our patient?

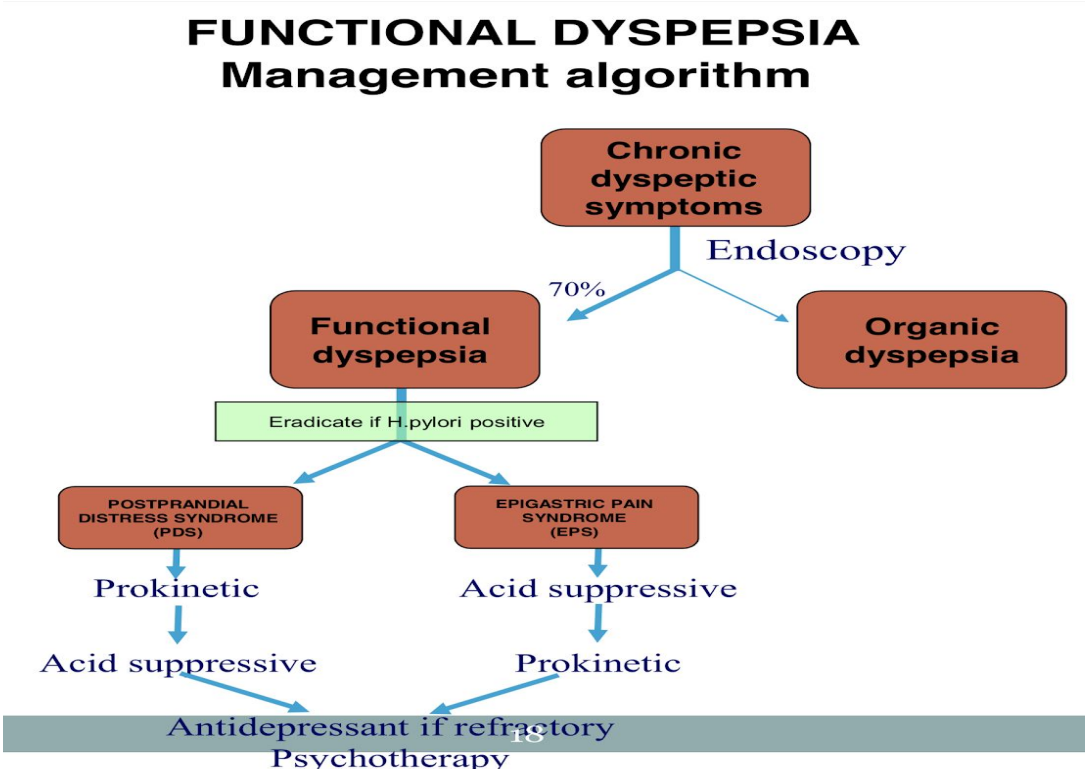
- Endoscopy (no alarming symptoms in this case so not the best answer)
- High dose PPI (if HP is negative)
- HP testing (the best answer according to this case, and in our culture H.pylori is prevalent so it is better to do it first)

* Drugs that might cause upper epigastric pain are NSAIDs, aspirin, antibiotics, iron and potassium supplements.

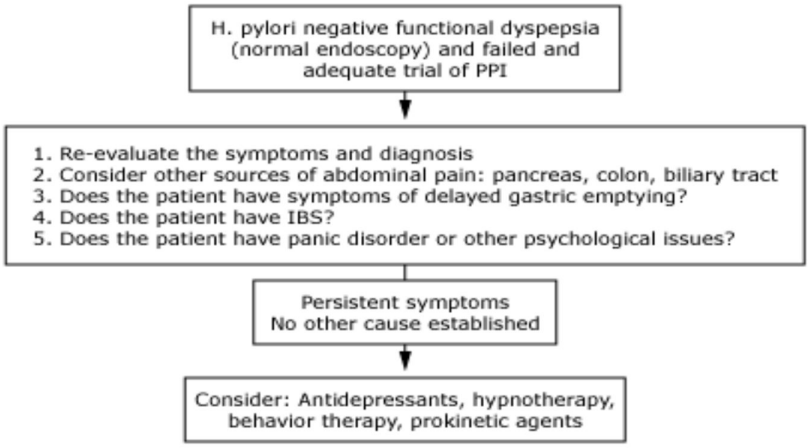
* When do we do endoscopy? In presence of alarming symptoms or when not responding to treatment or age >55.

The result in this case:

- HP serology was positive. Received eradication therapy.
- You treat H.Pylori but symptoms are constant?
- Test for HP eradication was negative

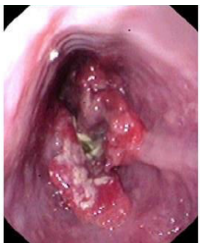


Management of Functional Dyspepsia:



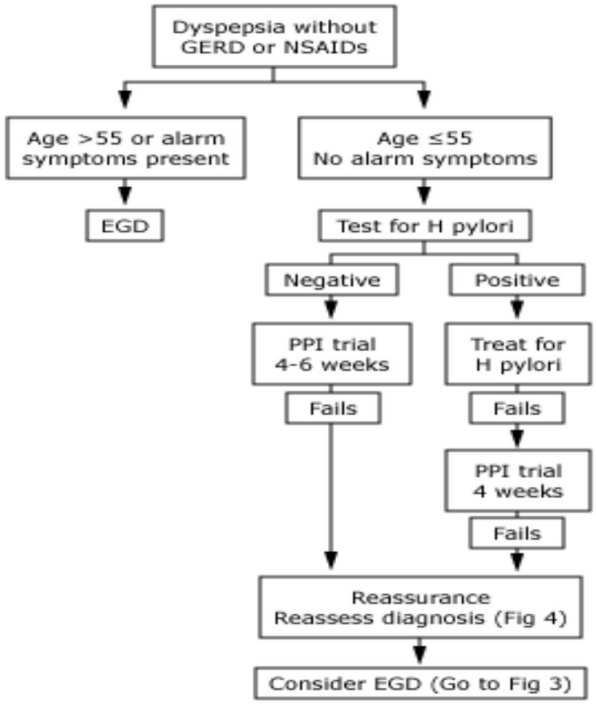
Case 2:

- Patient was a 60 y/o lady who was referred to me because of constant epigastric pain
- She mentioned 6 kg wt loss since 3 months ago She was anemic with ferritin = 5



What is the best diagnostic test? Endoscopy (presence of alarming symptoms in this case as weight loss and anemia)

Management of dyspepsia based on age and alarm features:

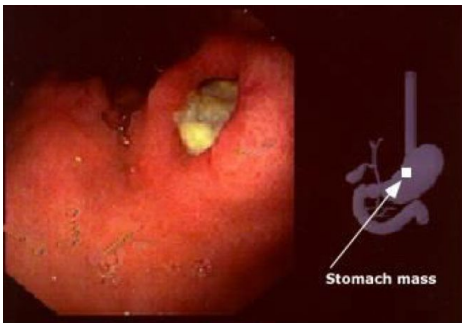


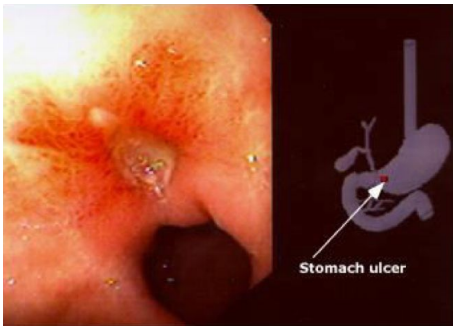
H.pylori

Case 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.
  We need to do an analysis first according to the algorithm , but in this case it's not straightforward since she is less than 55 y/o + chronic pain but recently she had melena (alarming symptom)

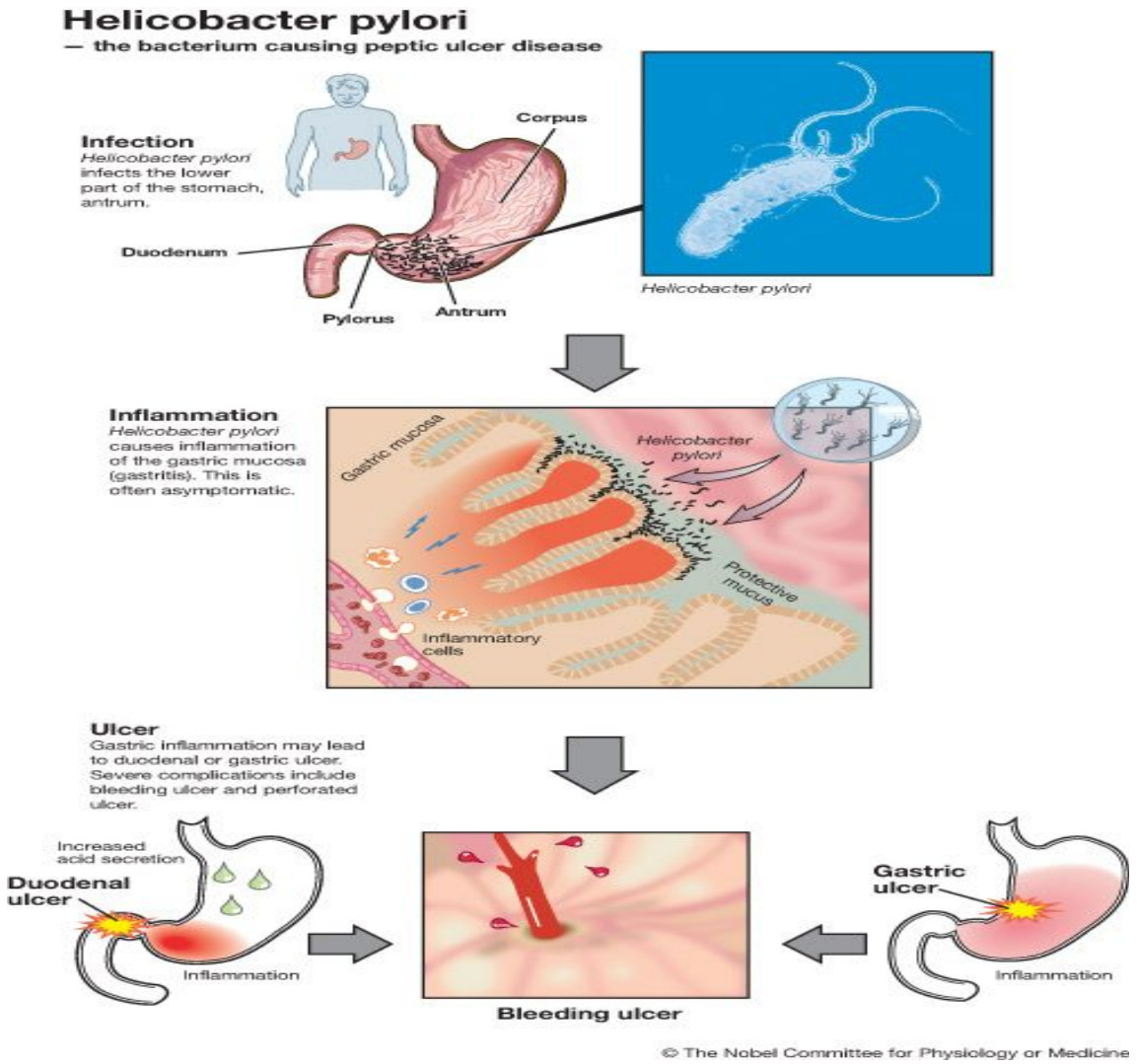
What is the next step? Endoscopy (it showed gastric ulcer caused by H.pylori)
What also increase risk of ulcer? Stress of burn, gastrinoma...etc





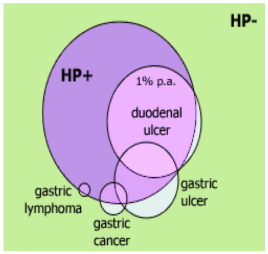
Helicobacter Pylori:

Helicobacter Pylori - Gram negative organism	
Characteristics	• Slow growing (culture takes 6-8 weeks) • Microaerophilic • Highly motile • Spiral • Urease producing (we detect it in breath test)
Transmission	Transmission occurs predominantly in children and socio-economic status of the family is the main risk factor as reflected by the level of sanitation and household hygiene. Route of transmission is from person to-person through: • Fecal-oral route • Gastro-oral route • Oral-oral route One of the commonest human infection



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.

H.pylori as a cause of PUD:



- 95% of duodenal ulcer is HP +ve
- 50% of HP patients are asymptomatic
- HP may cause ulcer or gastric cancer or gastric lymphoma

Diagnosis:

- **Non endoscopic methods:** serum antigen, UBT (urea breath test), stool antigen.
- **Endoscopic methods:** histology, rapid urease test (of biopsy)أسر عهم, culture, PCR

- Problem of serology essay is that if +ve we don't know is the patient treated or not (may be present after treatment)
- Urea breath test and stool antigen are good to measure effect of treatment
- Mechanism of detecting HP in breath test: urease in HP breaks the bound between urea and CO2 thus radiolabeled CO2 is absorbed by stomach then goes through blood stream into lungs (exhaled)
- HP +ve patients -> treat from HP even if asymptomatic

European Helicobacter Pylori Study Group Guidelines		
• Triple therapy with omeprazole (20 mg twice daily), amoxicillin (1g twice daily), and clarithromycin (500mg 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.		
Regimen all of them are 1st line therapy	**Comment**	
PPI, amoxicillin 1 gm, clarithromycin 500 mg all twice daily for 7-14 days - sometimes there is clarithromycin resistance	1st line treatment regimen of choice (can substitute metronidazole 500 mg twice daily for amoxicillin but only in penicillin allergic patients) - Metronidazole has bad taste	
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	
Bismuth 525 mg, metronidazole 500 mg, tetracycline 500 mg all four times daily with a PPI twice daily for 7-14 days	Can be used as 1st line treatment (7-14 days) but generally reserved for retreatment (14 days)	
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

- If all medications didn't work then do culture
- If you thought someone may have resistance to clarithromycin (±penicillin allergy) then you can go **directly to quadruple therapy**

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

Clarithromycin-resistant bacteria:

- A 10-day sequential regimen (a PPI and amoxicillin 1 g, each given twice daily for the first 5 days, followed by the PPI, clarithromycin 500 mg, and tinidazole 500 mg, each given twice daily for the remaining 5 days)
- 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:

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.

Failure of Therapy:

Poor compliance with medication, and patient demographics such as younger age, smoking, prior antibiotic use, and underlying condition (functional dyspepsia vs. peptic ulcer). Poor compliance is the most important limitation

- **Sequential therapy** (total 10 days) was made when some patients appears to have resistance for quadruple therapy. Sequential therapy is giving one antibiotic (amoxicillin + PPI) for 5 days then giving (clarithromycin + metronidazole + PPI) for the next 5 days

		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%

When some patients had resistance for **sequential therapy**, they came up with **rescue therapy**

Rescue Therapy	
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

If all medications listed above didn't work then we do culture Based on culture treatment. Why usually we don't do culture? because it takes time, we usually do UBT , endoscopy and routine histopathology	

Some patients don't continue their therapy course, why? 1- they think they are already cured (but actually it was PPI relief) 2- they want to stop because of therapy's side effects

Peptic Ulcer (Extra)

Peptic ulcer disease is a term applied for both duodenal ulcer and gastric ulcer.

Causes:

1. **Most common causes:**
- Helicobacter pylori infection.
- NSAIDs (2nd most common)—inhibit prostaglandin production which is the major stimulant for mucus production that form the protective barrier, leads to impaired mucosal defenses.
- Acid hypersecretory states, such as "Zollinger–Ellison syndrome" (3rd most common)

2. **Other causes:**
- Smoking—ulcers twice as likely in cigarette smokers as in nonsmokers.
- Alcohol and coffee—may exacerbate symptoms, but causal relationship as yet unproven.
- Other potential but unproven causes include emotional stress, personality type ("type A"), and dietary factors

Other causes: They don't cause the ulcer disease but they delay the healing and are associated with the development of gastritis.*Stress ulcer* which are caused by burns , head injury , its mechanism is that there is an intense vasoconstriction of the vasculature that supplies the gastric mucosa. Leading to sloughing of these cells and ulceration.

Clinical Features:

1. **Epigastric pain:**
- Sharp, dull, Aching in nature.
- Gnawing Burning epigastric pain or "hungry" feeling.
- Nocturnal symptoms and the effect of food on symptoms are variable.
- Relieved by milk, food, or antacids.
- Epigastric tenderness

➢ Duodenal ulcer : the pain relieved by food + weight gain.
➢ Gastric ulcer : the pain exacerbated by food + weight loss.

2. **May be complicated by upper GI bleeding.**
3. **Other symptoms: nausea/vomiting, early satiety, and weight loss.**

TABLE 3-4 Duodenal Versus Gastric Ulcers

	Duodenal Ulcers	Gastric Ulcers
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)
***Helicobacter pylori* infection**	70% to 90% of patients	60% to 70% of patients
Malignant potential	Low (malignancy is very rare) should undergo biopsy to rule out	High (5% to 10% are malignant)—malignancy
Location	Majority are 1–2 cm distal to pylorus (usually on posterior wall)	Type I (most common, 70%): on lesser curvature Type II: gastric and duodenal ulcer Type III: prepyloric (within 2 cm of pylorus) Type IV: near esophagogastric junction
Age distribution	Occurs in younger patients (<40)	Occurs in older patients (>40)
Associated blood type	Type O	Type A
Risk factors	NSAIDs	Smoking
Other	Eating usually relieves pain Nocturnal pain is more common than in gastric ulcers	Eating does not usually relieve pain Complication rates are higher than those of duodenal ulcers. There is a higher recurrence rate with medical therapy alone

Complications:

Symptoms that suggest complications related to a peptic ulcer include:

- The sudden development of severe, diffuse abdominal pain may indicate perforation.
- Vomiting is the cardinal feature present in most cases of pyloric outlet obstruction.
- Hemorrhage may be heralded by nausea, hematemesis, melena, or dizziness.

TABLE 3-6 Complications of Peptic Ulcer Disease

	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 test 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 a nasogastric tube, add 750 mL saline, aspirate after 30 min—test is positive if aspirate >400 mL)	Initially, nasogastric suction; replace electrolyte/volume deficits; supplement nutrition if obstruction is longstanding 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 ≥6 units of blood	**Peptic ulcer disease is the most common cause of upper GI bleeding**

Warning sign of peptic ulcers → Compilation:

- Sudden and severe, diffuse abdominal pain → **perforation**.
- Vomiting "Is the cardinal feature in"→ **pyloric outlet obstruction.**
- Nausea, hematemesis, melena, or dizziness. → **Hemorrhage**.

Diagnosis:

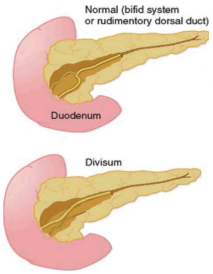
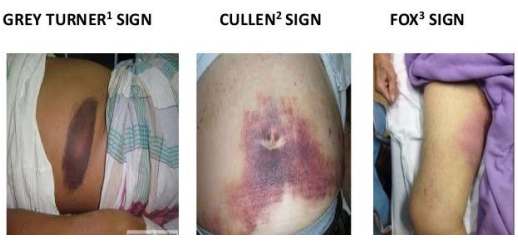
Endoscopy (most accurate test)

	- Essential in diagnosis of gastric ulcers because biopsy is necessary to rule out malignancy—duodenal ulcers do not require biopsy. **Barium swallow:** - Sometimes used initially but is less reliable than endoscopy. - Double-contrast techniques preferred due to improved accuracy. **Laboratory test**: - for diagnosis of H. pylori infection. - Biopsy: (gold standard). - Urease detection via urea breath test is (The most convenient test) (sensitivity and specificity >95%). - Serology (lower specificity). **Serum gastrin measurement**—if considering Zollinger–Ellison syndrome as a diagnosis.
Treatment:	**Medical:** • **Supportive (patient directives):** - Discontinue aspirin/NSAIDs. - Restrict alcohol, Stop smoking, decrease emotional stress. - Avoid eating before bedtime and decrease coffee intake. • **Acid suppression:** - H2 blockers "accelerate healing of ulcer" - PPIs. **(most effective)** - Antacids. "Symptomatic relief" • **Eradicate H. pylori:** - With triple or quadruple therapy (PPI, bismuth and 2 antibiotics). • **Cytoprotection:** - Sucralfate. "facilitates ulcer healing." - Misoprostol. "Can cause GI upset (common side effect)" • **Treatment regimens:** - If H. pylori test is positive, begin eradication therapy. - Also begin acid-suppression with antacids, an H2 blocker, or a PPI. - If the patient has an active NSAID-induced ulcer, stop NSAID use . **Surgical:** - Rarely needed and required for the complications of PUD (bleeding, perforation, gastric outlet obstruction)

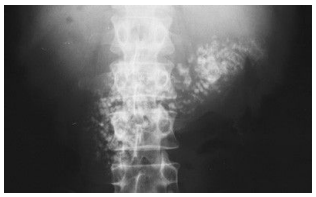
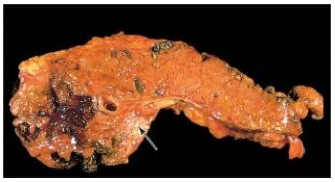
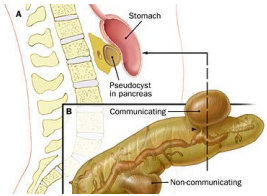
TABLE 3-5 *Helicobacter pylori* Eradication

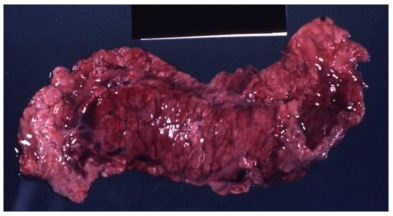
	Regimen	Advantage	Disadvantage
Triple therapy	PPI plus two antibiotics	Twice daily dosing	More expensive than bismuth-based triple therapy
Quadruple therapy	PPI, bismuth subsalicylate, and two antibiotics	Half the time as triple therapy (a 1-week program as opposed to 2 weeks for triple therapy), yet reaps similar eradication results	Expense of PPI

Acute pancreatitis (Extra)

General characteristics:	• 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: 1. **Mild acute pancreatitis** is most common and responds well to supportive treatment. 2. **Severe acute pancreatitis** (necrotizing pancreatitis) has significant morbidity and mortality.
Causes:	1. **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" 2. **Alcohol abuse (40%)** 3. Post-ERCP[1]—Pancreatitis occurs in up to 10% of patients undergoing ERCP[2]. 4. Viral infections (e.g., mumps, Coxsackievirus B) 5. Drugs: Sulfonamides, thiazide **diuretics,NSAIDS**, furosemide, estrogens, HIV medications. 6. Postoperative complications. (high mortality rate) 7. Scorpion bites. 8. Pancreas divisum[3]. "Pic" 9. Pancreatic cancer. 10. Hypertriglyceridemia, hypercalcemia. 11. Uremia. 12. Blunt abdominal trauma **(most common cause of pancreatitis in children)** 
Clinical Feature:	**Symptoms:** 1. 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. 

[1] **"Endoscopic retrograde cholangiopancreatography"** Read
[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.

	- *Turner's sign:* Bluish purple discoloration of flanks -> tissue catabolism of Hb.
Diagnosis:	**<u>Laboratory studies:</u>** • **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** **G**lucose >200 mg/dL — Calcium <8 mg/dL; Decrease in **H**ematocrit >10% — <3 criteria—1% **A**ge >55 years — Pa_{O_2} <60 mm Hg — 3–4 criteria—15% **L**DH >350 — **B**UN increase >8 mg/dL — 5–6 criteria—40% **A**ST >250 — **B**ase deficit >4 mg/dL — >7 criteria—100% **W**BC >16,000 — Fluid sequestration >6 L **<u>Abdominal radiograph:</u>** • 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. "Pic"  **<u>Abdominal ultrasound:</u>** • Can help in identifying cause of pancreatitis (e.g., gallstones). • Useful for following up pseudocysts or abscesses. **<u>CT scan of the abdomen:</u>** • **(Most accurate test)** "for diagnosis of acute pancreatitis and for identifying complications of the disease." • Indicated in patients with severe acute pancreatitis. **<u>ERCP (indications):</u>** • Severe gallstone pancreatitis with biliary obstruction. • To identify uncommon causes of acute pancreatitis if disease is recurrent.
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 in multiple 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"

Case 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 We get from the history : Age (not old), chronicity and no alarming symptoms.

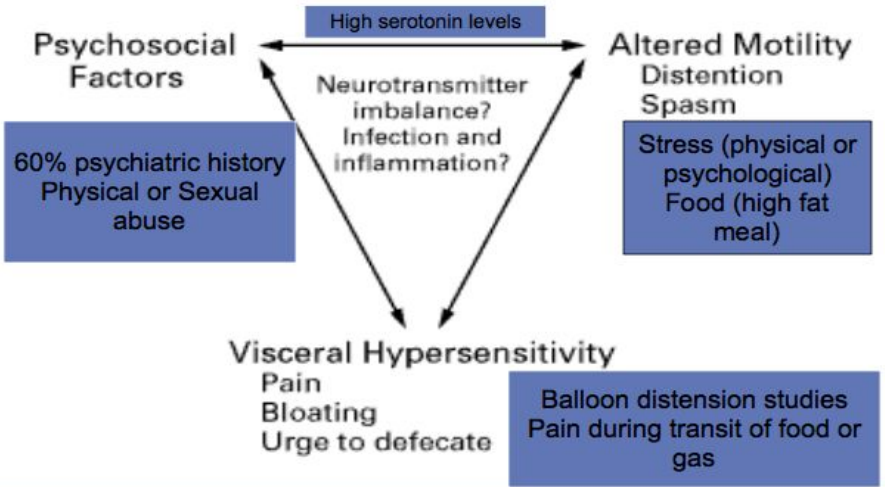
What is the likely diagnosis? IBS

Irritable bowel syndrome

- 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.
- *IBS refers to an idiopathic disorder associated with an intrinsic bowel motility dysfunction (abnormal resting activity of GI tract) that affects 10% to 15% of all adults.

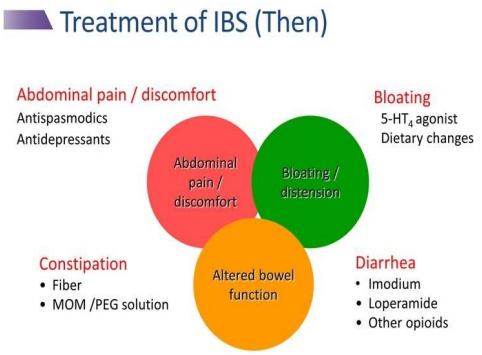
- Is predominantly a pain syndrome of unknown etiology that is often relieved by bowel movement.
- Educate patient that this is a chronic disease that stays for life

Introduction	• First described in 1771. • 50% of patients present <35 years old. • 70% of sufferers are symptom free after 5 years. • GPs will diagnose one new case per week. • GPs will see 4-5 patients a week with IBS. • Point prevalence of 40-50 patients per 2000 patients. غالبا عند الناس الأغنياء أكثر من الفقراء
General characteristics	• IBS is a chronic continuous or remittent functional GI illness. • It has no recognized organic disease and has no specific cause. • 50% of referrals to gastroenterologist. • Common associated findings include depression, anxiety, and somatization. Psychiatric symptoms often precede bowel symptoms. Symptoms are exacerbated by stress and irritants in the intestinal lumen. • All laboratory test results are **normal,** and no mucosal lesions are found on sigmoidoscopy. "IBS is a benign condition and has a favorable long-term prognosis." • Symptoms should be present for at least 3 months to diagnose IBS.
Causes	• The cause of IBS is incompletely understood but biopsychosocial factors are thought to play an important role, along with luminal factors, such as diet and the gut microbiota.
Clinical Feature	• **Recurrent Cramping abdominal pain characterized by:** "Location varies widely, but **sigmoid colon** is the common location of pain" ❑ Relieved by defecation ❑ Less at night. • Change frequency and consistency of stool Such as diarrhea , constipation or alternating diarrhea ,and constipation. "20% of IBS have constipation only. The majority are diarrhea or alternating." • Abnormal stool passage (straining, urgency or feeling of incomplete evacuation). • Bloating or feeling of Abdominal distention.
Associated Symptoms	• In people with IBS in hospital OPD. ❖ 25% have depression. ❖ 25% have anxiety. • In one study30 % of women IBS sufferers have fibromyalgia.
Epidemiology	It is estimated that between 10% and 15% of the population of North America, or approximately 45 million people, have irritable bowel syndrome. Gender differences: - Affects up to 20% of adults (70% of them are **women**). IBS tends to be more common in In women, IBS is 2 to 3 times more common than in men. Age: - **Young**. - High prevalence of psychiatric disorders (anxiety and depression were the most common). Only 25% of people with this condition seek medical care. only about 30% of them will consult a doctor about their symptoms

Pathophysiology	 • Heredity; Nature vs Nurture • Dysmotility, "Spasm" • Visceral Hypersensitivity • Altered CNS perception of visceral events • Psychopathology • Infection/Inflammation • Altered gut flora
Diagnosis	• This is a clinical diagnosis, and a **diagnosis of exclusion.** • Initial tests that may help exclude other causes include CBC, renal panel, fecal occult blood test, stool examination for ova and parasites, ESR and possibly a flexible sigmoidoscopy. • You must first exclude lactose intolerance , IBD , celiac disease , carcinoid, Giardia infection and anatomic defects of bowel as the cause. • Rome III Diagnostic criteria. **By Rome III criteria : Recurrent abdominal pain or discomfort > or = 3 days per month in last 3 months:We don't use Rome III any more because the sensitivity is low but it's important to know it** • **At least 12 weeks history** Either continuous or intermittent **, which need not be consecutive in the last 12 months of abdominal discomfort or pain that has 2 or more of the following:** 1. Pain or discomfort **improve with defecation**. 2. Symptoms onset is associated with change in **frequency of stool**. 3. Symptoms onset is associated with change in **form (appearance) of stool.** **22.83 Rome III criteria for diagnosis of irritable bowel syndrome** Recurrent abdominal pain or discomfort at least 3 days/mth in the last 3 months, associated with *two or more* of the following: • Improvement with defecation • Onset associated with a change in frequency of stool • Onset associated with a change in form (appearance) of stool • Rome III Diagnostic Criteria in general: ❖ Feeling of incomplete evacuation. ❖ Passing mucus per rectum (not an alarming symptom) ❖ Abdominal fullness, bloating or swelling.

	Since it's syndrome it's associated with other conditions like anxiety and depression so don't forget to ask about them and patients sleeping patterns • Manning's Criteria. • Rome II Diagnostic Criteria ❖ Supportive symptoms. ➔ Constipation predominant: one or more of: ➢ BO less than 3 times a week. ➢ Hard or lumpy stools. ➢ Straining during a bowel movement. ➔ Diarrhoea predominant: one or more of: ➢ More than 3 bowel movements per day. ➢ Loose [mushy] or watery stools. ➢ Urgency. **22.84 Supporting diagnostic features and alarm features in IBS** **Features supporting a diagnosis of IBS** • Symptoms > 6 mths • Frequent consultations for non-gastrointestinal problems • Previous medically unexplained symptoms • Stress worsens symptoms **Alarm features** • Age > 50 yrs; male gender • Weight loss • Nocturnal symptoms • Family history of colon cancer • Anaemia • Rectal bleeding **Ask about Alarm symptoms that suggest other serious diseases. (to exclude them):** ❑ PR bleeding , Weight loss. ❑ Family history of cancer , Onset >45 years of age. ❑ Fever, Anemia. ❑ Progressive deterioration, Steatorrhea and dehydration. • A firm diagnosis of IBS based on validated HX, and a normal physical examination, coupled with limited relevant diagnostic testing is reassuring to patients. • Endoscopy? Only if you want to roll out other diseases **22.83 Rome III criteria for diagnosis of irritable bowel syndrome** Recurrent abdominal pain or discomfort at least 3 days/mth in the last 3 months, associated with *two or more* of the following: • Improvement with defecation • Onset associated with a change in frequency of stool • Onset associated with a change in form (appearance) of stool
Diagnostic Testing in IBS	• CBC, LFT • Stool analysis • TFT • Celiac Ab • Summary of the use of blood tests for the diagnosis of IBS: 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 We order it if the symptoms is affected by dietary intake
Abdominal Imaging	• US • Barium enema • Colonoscopy • prevalence of colorectal cancer in these studies was low, ranging from 0 to 0.51%. In IBS usually all imaging appears normal
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.
Management and treatment:	• Patients' concerns. (patient reassurance is very important… show him that you understand their complaints so they don't think have something serious) • Explanation. • Treatment approaches. Since it is multifactorial and we don't have treatment for everything , psychological support is important and it includes the acknowledgement of patient symptoms • Placebo effect of up to 70% in all IBS treatments. Because psychological effect plays a role in the symtoms • Treatment should depend on symptom sub-type. • Usually, no specific treatment is necessary. Manage the symptoms below as indicated: *"Anti Motility"* ➢ **Diarrhea**—diphenoxylate, *loperamide.* ➔ Increasing dietary fibre is sensible advice.While in constipation it's non dietary ➔ 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 for ischemic colitis compared [Ford et al. 2008 ➢ **Constipation**—Colace, psyllium, *cisapride.* ➔ Lifestyle Modifications ➔ Bowel Training and Education ➔ Fibre (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 fiber work as a sponge and make stool bulky) ➔ 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

Toskes et al. 1993; Jalihal and Kurian, 1990; Prior and Whorwell, 1987; Longstreth et al. 1981].

- ➔ 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
- ➔ 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%

- Why do we work on 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

- Avoid dairy products, avoid excessive caffeine.
- Tegaserod maleate (Zelnorm) is a serotonin agonist recently introduced for the treatment of IBS. "In a short-term study, it improved abdominal pain, bloating, and constipation in women."
- Fiber and diet.
- Pain Predominant. Antispasmodic agent (hyoscyamine or dicyclomine).
  - ❖ Antispasmodics will help 66%.
  - ❖ Mebeverine is probably first choice.
  - ❖ Hyoscine 10mg qid can be added

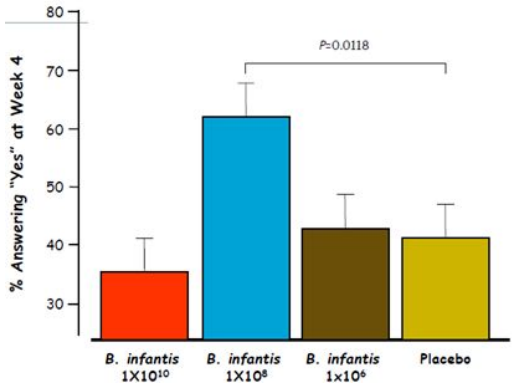
A recent meta-analysis of 22 studies involving 1778 patients and 12 different antispasmodic agents demonstrated modest improvements in global IBS symptoms and abdominal pain. However, up to 68% of patients suffered side effects when given the high dose required to improve abdominal pain

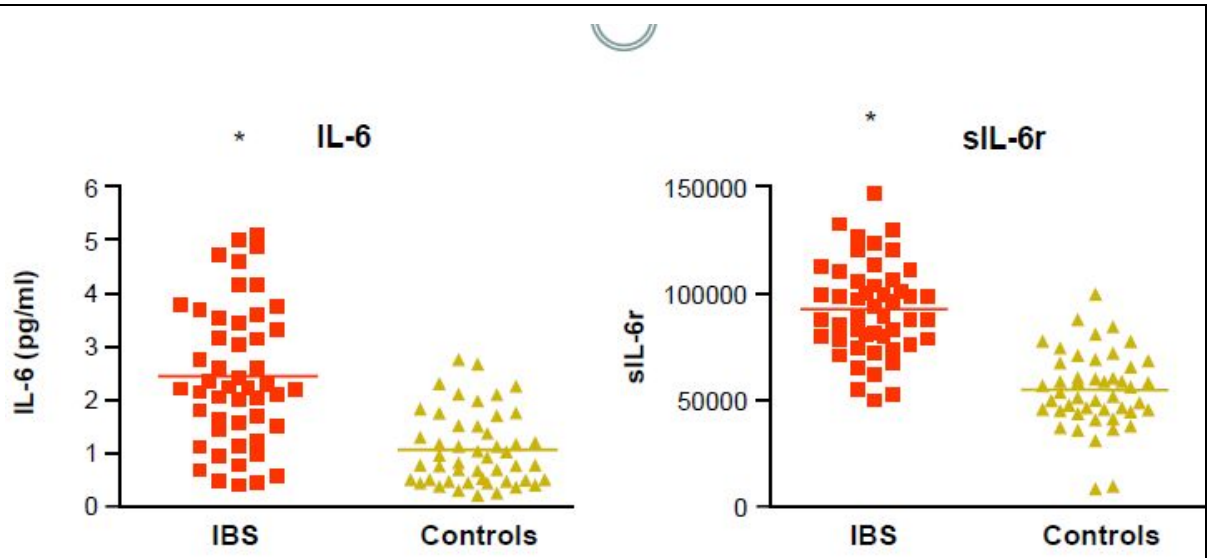
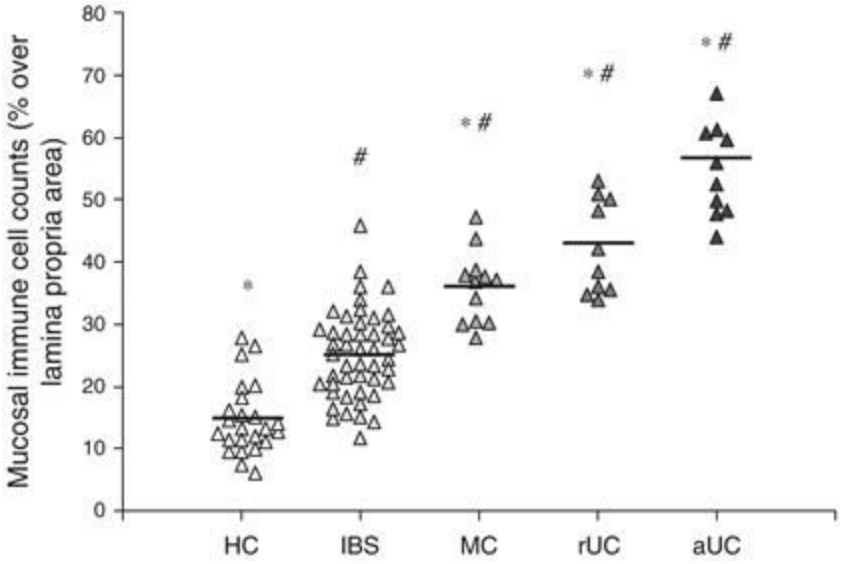
Page and Dirnberger, 1981

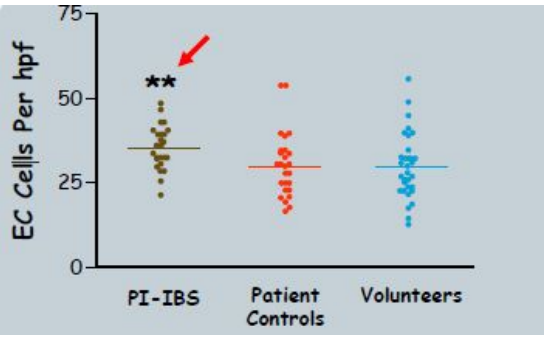
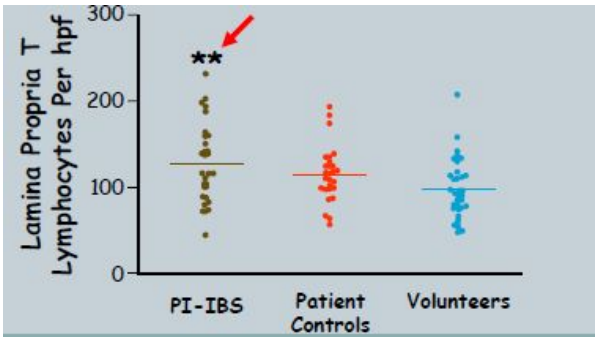
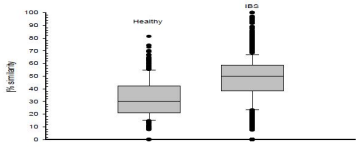
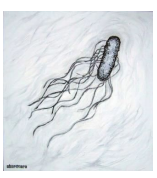
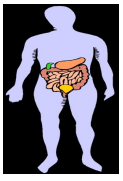
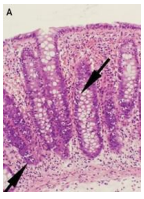
- Tricyclic and antidepressant (Resistant cases)
  - ❖ Poor evidence for efficacy.
  - ❖ Better evidence for tricyclics. because they have anti-cholinergic effect
  - ❖ Very little evidence for SSRIs:
    - ➔ six studies have been conducted to date, two each involving fluoxetine, paroxetine and citalopram

      Talley et al. 2008; Tack et al. 2006; Vahedi et al. 2005; Tabas et al. 2004; Kuiken et al. 2003; Masand et al. 2002].
    - ➔ 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
- **Probiotics** (live microbiota)
  - ❖ **Mode of Action of Probiotics?**
    - ➢ **Competition with, and exclusion, of pathogens**
    - ➢ **Anti-bacterial:**
      - ➔ **Produce bacteriocins**
      - ➔ **Destroy toxins**
    - ➢ **Enhance barrier function, motility**
    - ➢ **Enhance host immunity**
      - ➔ **Immune modulation**
      - ➔ **Cytokine modulation**
      - ➔ **IgA production**
    - ➢ **Metabolic functions**

sometimes probiotics aren't effective themselves but it's a placebo effect

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.
Global Assessment of Symptom Relief **Male's doctor skipped it**	
Alternative and Complementary Medicine	• Peppermint, germanium, lavender oils • RCTof 57 IBS patients randomized to receive either peppermint capsules or placebo demonstrated a significant benefit for the peppermint-treated group after 4 weeks. (ورق النعناع peppermint decrease spasms in bowel) • Seventy-five percent of the study group versus 38% of the placebo group reported a greater than 50% reduction in total IBS symptoms Cappello et al. 2007]. • 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 **Male's doctor skipped it**	• IBS network, • IBS support • Group Awareness

Systemic Immune Compartment in IBS Serum Cytokines **Male's doctor skipped it** **Female's doctor didn't skip**	
Mucosal Compartment **Male's doctor skipped it**	• Frank inflammation • Immune Activation ❖ ↑ IEL's ❖ ↑ CD3+, CD25+ Chadwick et al, 2002 • Decreased IgA+ B Cells Forshammar et al, 2008 • Altered expression of genes involved in mucosal immunity Aerssens et al, 2008 
Evidence for a role for the Gut Flora in IBS	• Direct evidence of an altered gut flora: ❖ Post-Infectious IBS (PI-IBS) ❖ Small Intestinal Bacterial Overgrowth (SIBO) ❖ Altered Colonic Flora • Evidence of physiological effects of an altered flora:

	❖ Changes in stool volume/consistency ➔ Bile salt deconjugation ❖ Alterations in gas volume/composition ➔ Fermentation • Mediator of pro-inflammatory state • Therapeutic impact of altering flora
Post-Infectious IBS	• **Patient say to you they had gastroenteritis and after it they started feeling IBS symptoms (even after a year from gastroenteritis they still get IBS symptoms)** • **10-14% incidence following confirmed bacterial gastroenteritis** **Dunlop, et al. 2003.** **Mearin, et al. 2005.** • **Risk factors** ❖ **Female** ❖ **Severe illness** ❖ **Pre-morbid psyche** ➔ **Depression** ❖ **Persistent inflammation** ➔ **EC cells** ➔ **T lymphocytes**  
Variability in Flora	 Codling et al, 2010 **Disturbed Flora** **Susceptible Host** **Inflammatory Response** **(e.g. after taking antibiotics)**  >  >  **>Myo-Neural Dysfunction >SYMPTOMS**

Subtypes	• **Diarrhoea predominant.** • **Constipation predominant.** • **Pain predominant.** Some patients have all of them which are very difficult to treat
Differential Diagnosis	• **Inflammatory bowel disease.** • **Cancer.** • **Diverticulosis.** • **Endometriosis.** • **Celiac disease**
Assessment	• **Results should be normal or nonspecific.** • **Abdomen and rectal examination.** Complete history is important We don't take biopsy for IBS but if done it will be normal

Abdominal Pain Clinical Pearls:

- Pain awakening the patient from sleep should always be considered significant.
- Pain almost always precedes vomiting in surgical causes; converse is true for most gastroenteritis and NSAP (non-specific abdominal pain).
- Exclude life threatening pathology.
- BHCG (Beta Human chorionic gonadotropin) in female of childbearing age.
- Initial workup of chronic abdominal pain should be focused on differentiating benign functional illness from organic pathology.
- Features that suggest organic illness include unstable vital signs, weight loss, fever, dehydration, electrolyte abnormalities, symptoms or signs of gastrointestinal blood loss, anemia, or signs of malnutrition.