## 1 PEPTIC ULCER DISEASE (PUD)

- PUD is characterized by mucosal damage secondary to pepsin & gastric acid secretion
- It is the principal cause of upper gastrointestinal (UGI) hemorrhage
- Most commonly occurs in the stomach & proximal duodenum; infrequently, in the lower esophagus, distal duodenum or jejunum
- Giant ulcer w/ size of 3 cm, is an atypical type of PUD that is now rarely encountered
- Refractory PUD is considered in patents w/ ulcer that failed to heal after 8-12 wk of therapy

## 2 DIAGNOSIS

- Usually based on clinical features & specific testing, however, signs &symptoms are nonspecific

**History**

**Clinical Features**

- Epigastric pain is the most common symptom of PUD but occurs only in minority of patients
  - Pain of duodenal ulcer usually occurs 2-3 hr after a meal, improves w/ food or antacid, & sometimes awakens patient at night
  - Pain of gastric ulcer is more commonly worsened by food intake
    - May be associated w/ weight loss due to fear of food intake
- Other symptoms include indigestion, vomiting, loss of appetite, inability to tolerate fatty foods, heart burn
  - Nausea & vomiting is commonly experienced by patients w/ prepyloric or pyloric channel ulcers

**Risk Factors**

- In 70% of cases, 25-64 yr old patients are affected
- 48 & 24% of cases are secondary to *Helicobacter pylori* infection & nonsteroidal anti-inflammatory drugs (NSAIDs), respectively
  - NSAIDs inhibit the formation of prostaglandins & their protective effect on the gastric mucosa (ie stimulates mucus & bicarbonate secretion & epithelial cell proliferation, & increase of mucosal blood flow)
    - Patients who are on long-term NSAIDs have an annual risk of life-threatening ulcer-related complication of 1-4%
  - *H. pylori* plus NSAID use increases the risk & intensity of NSAID-related mucosal damage
- Other causes may include use of steroids, bisphosphonates, potassium chloride or chemotherapeutic agents, presence of acid-hypersecretory states (eg Zollinger-Ellison syndrome), cancer or stress [eg multiorgan failure, ventilator support, extensive burns (Curling's ulcer) or head injury (Cushing's ulcer)]
- In patients w/ NSAID-related GI complications, additional contributing factors include history of complicated GI event, age, concomitant use of anticoagulants, corticosteroids, other NSAIDs including low-dose Aspirin, high-dose therapy, chronic debilitating disorders

**Alarm Symptoms**

- Hematemesis or melena may suggest bleeding
- Vomiting may be due to obstruction
- Anorexia or weight loss may suggest cancer
- Upper abdominal pain radiating to the back that persists may be due to penetration
- Spreading upper abdominal pain that is severe may suggest perforation

**Physical Exam**

- Typically indistinct esp in patients w/ uncomplicated PUD
  - Most patients may only have mild epigastric tenderness
- Acute abdomen may be present in patients w/ perforation
- Anemia may be observed in patients w/ hemorrhage

**Diagnostic Procedures**

**Esophagogastroduodenoscopy (EGD)**

- Identifies gastric & duodenal ulcers & cancers w/ 90% sensitivity & specificity
  - NSAID-associated lesions typically presents w/ shallow flat antral ulcer w/ associated lesions
- Recommended in patients w/ evidence of bleeding, weight loss, chronic PUD, persistent vomiting or any alarm features that may suggest significant structural disease or malignancy, in patients whose symptoms do not respond to pharmacotherapy, & in >50 yr old patients w/ new-onset dyspepsia
- Main role in uncomplicated PUD is to confirm the diagnosis & to rule out cancer
- May be used for surveillance of ulcers
  - Should be considered in patients w/ duodenal ulcers who have persistent symptoms despite an appropriate therapy
    - May rule out refractory peptic ulcers & ulcers w/ nonpeptic origin
    - Has low yield if patients' symptoms resolved after course of acid suppression w/ eradication treatment for *H pylori,* & discontinuation of NSAIDs
  - May identify gastric cancer early in patients w/ gastric ulcer, hence improving survival
    - Should be performed depending on patients risk for gastric ulcer
    - Should be considered in patients w/ gastric ulcer w/o clear etiology & in those who did not undergo biopsy during index EGD
  - Should be performed in patients w/ refractory PUD until the ulcer has healed or the etiology has been identified
- Allows biopsy of gastric lesion
  - Indicated in gastric ulcer w/ features of cancer (ie associated mass lesion, elevated irregular ulcer borders, & abnormal adjacent mucosal folds)
  - At first , some malignant ulcers may appear endoscopically benign
    - 2-5% of malignant ulcers may have false-negative biopsy results
    - Ulcers that are not healed after 8-12 wk of medical therapy should have a repeat biopsy
  - Routine cytologic brushings may add to the sensitivity but is not advised as an alternative or adjunct to endoscopic biopsy

## 2 DIAGNOSIS (CONT'D)

**Diagnostic Procedures (Cont'd)**

**Esophagogastroduodenoscopy (Cont'd)**

- May also be used in diagnosing, prognosticating, & managing complications of PUD
  - In bleeding PUD, EGD done w/in 24 hr of admission has been shown to reduce the need of blood transfusion, shorten intensive care unit (ICU) & hospital stays, decrease need for surgery, lowers mortality rate
    - Patients w/ features of high risk of rebleeding on endoscopy (ie presence of adherent clots, visible vessels, active arterial bleeding) should undergo endoscopic therapy to attain hemostasis & to lower the risk of rebleeding
    - Repeat endoscopic therapy is advised prior to considering surgical or radiological intervention in patients who rebleed after initial endoscopic therapy
  - Allows identification of ulcer penetration to adjacent organs like liver & spleen through biopsy obtained via endoscopy
  - Important in confirming the presence & distinguishing benign from malignant obstruction
    - Endoscopic ballon dilation has been used to manage benign gastric outlet obstruction causing good to excellent short-term relief of symptoms in 67-83% of patients
- Contraindicated in patients w/ acute perforated peptic ulcer
  - In some studies, role has been limited in identifying perforation site & in guiding subsequent laparoscopic intercorporeal suture repair w/ omental patch
- Histological exam, culture, or rapid urease testing of *H pylori* may also be done endoscopically
  - Please see *Helicobacter pylori* Infection Disease Management Chart for details

**Radiologic Upper Gastrointestinal Series**

- May be an option when EGD is not available
- Not effective in identifying ulcers of <0.5 cm in size & does not allow biopsy

**Lab Tests**

- W/ minimal role in evaluating patients w/ uncomplicated PUD
- Fasting serum gastrin level may be done in patients w/ duodenal ulcer in whom presence of other diagnosis (eg Zollinger-Ellison syndrome) is suspected
  - Please see Zollinger-Ellison Syndrome Disease Management Chart for details
- *H pylori* testing is important to diagnose & manage concomitant infection which may include serologic ELISA, urea breath test or stool antigen test
  - Please see *Helicobacter pylori* Infection Disease Management Chart for details

## A NONPHARMACOLOGICAL THERAPY

**Patient Education**

**Alcohol**

- Alcohol is a strong promoter of acid secretion
- Also an independent risk factor for PUD
  - Chronic alcohol drinkers develop ulceration while occassional drinker may only have gastritis
- Recurrence rates of patients who consume alcohol were significantly higher in patients w/ gastric ulcer recurrence compared to duodenal ulcer recurrence w/ ulcers reappearing at the same or adjacent sites as the previous ulcers

**Diet**

- There is limited or no evidence that changing the diet fastens ulcer healing or prevents recurrence
- Patient should be advised to avoid specific foods that may precipitate dyspepsia
  - Milk has been shown to be a potent gastric acid secretion stimulus

**Medication Use**

- Aspirin, NSAIDs & corticosteroids can cause peptic ulceration
- Patient should be advised to discontinue NSAID use; if not possible, alternative methods may be considered to prevent development of peptic ulceration & mucosal injury:
  - NSAID should be given w/ proton pump inhibitor (PPI), high-dose (2x) histamine$_2$-receptor antagonist ($H_2$RAs), or Misoprostol, *or*
    - Please see next page for details
  - Selective cyclooxygenase 2 (COX-2) inhibitor should be substituted for a traditional NSAID
    - COX-2 inhibitors causes significantly lower incidence of ulceration & ulcer complications
    - Usefulness has been decreased due to their association w/ myocardial infarction & thrombotic cardiovascular events
    - Current studies suggests that both coxibs & NSAIDs, w/ possible exception of full-dose Naproxen, increase CV risk

## A NONPHARMACOLOGICAL THERAPY (CONT'D)

**Patient Education (Cont'd)**

**Smoking**

- Smoking increases the risk of ulcer recurrence & slows healing
  - Recurrence rates of patients who smoked were significantly higher in patients w/ gastric ulcer recurrence compared to duodenal ulcer recurrence w/ ulcers reappearing at the same or adjacent sites as the previous ulcers
  - Risk of PUD is correlated w/ the number of cigarettes smoked per day
- Patient should be advised to stop smoking

## B PHARMACOLOGICAL THERAPY

- The most appropriate therapy for PUD depends on the cause
- Treatment of both gastric & duodenal ulcers involves suppression of acid secretion, eradication of *H pylori* (if present) & avoidance of NSAIDs
  - Antisecretory therapy speeds up the healing process & allows faster relief of symptoms
  - Eradication of *H pylori* in high-risk patients has been shown to greatly lower the risk of subsequent ulceration
- It is important to suppress nocturnal acid secretion in patients w/ duodenal ulcers
- Maintenance antisecretory therapy may be recommended to patients w/ high-risk for ulceration (ie history of ulcer complications, has frequent recurrences)

**Antacids**

- Neutralizes gastric acid & lowers pepsin activity
- Effective in relieving symptoms of PUD, promoting healing of ulcers & reducing recurrence
- Used only for short-term relief of symptoms
- May be absorbable (eg Sodium bicarbonate, Calcium bicarbonate) or non-absorbable (eg Aluminum or Magnesium hydroxide)
  - Absorbable antacids may provide fast & complete neutralization but may cause alkalosis & should be used only for 1-2 days
  - Non-absorbable antacids are preferred due to lesser systemic side effects

**Bismuth subcitrate**

- Has high affinity for damaged tissue
- Coats the base of the ulcer crater that provides protection against gastric acid, pepsin & bile
- Treatment efficacy is comparable w/ $H_2$RAs & other ulcer healing agents

**Histamine$_2$-Receptor Antagonist ($H_2$RAs)**

- Eg Cimetidine, Ranitidine, Famotidine, Nizatidine
- Decreases gastric secretion by blocking histamine action at the $H_2$-receptors in the parietal cells of the stomach
- All $H_2$RAs are equally effective in healing duodenal ulcers
  - Healing rates were 70-80% after 4 wk of therapy & 87-95% after 8 wk
- Double-dose of $H_2$RAs are effective in decreasing the risk of NSAID-induced endoscopic gastric ulcer
  - $H_2$RAs given w/ NSAIDs may be a cost-effective way of preventing ulcer bleeding secondary to NSAID use, however, no clinical data is available that proves that this strategy prevents ulcer complication
  - No studies have evaluated the efficacy of $H_2$RAs in chronic NSAID users
  - Less effective than PPIs
- $H_2$RAs are generally well tolerated but can cause mild central nervous system effects due to their ability to cross the blood-brain barrier & react w/ CNS histamine receptors
  - Cimetidine & Ranitidine both interact w/ hepatic cytochrome P-450 mixed oxidase system, hence can alter the metabolism of different drugs

## B PHARMACOLOGICAL THERAPY (CONT'D)

**Proton Pump Inhibitors (PPIs)**

- Eg Pantoprazole,Omeprazole, Esomeprazole, Lansoprazole, Rabeprazole
- Most potent inhibitor of gastric acid secretion
- Inhibits the activity of $H^+/K^+$-ATPase of the gastric parietal cells
- Has similar safety profile as w/ $H_2$RAs, but PPIs heal peptic ulcers faster than $H_2$RAs
  - Has 80-100% healing rate after 4 wk of therapy in patients w/ duodenal ulcer & 8 wk for patients w/ gastric ulcer
- Extensively used in combination w/ NSAID to prevent NSAID-induced peptic ulcer
  - Associated w/ significant risk reduction for upper GI bleeding in patient who uses NSAIDs
  - Main agent for prophylaxis & treatment of NSAID-related upper GI injury
- May also be used in patients w/ gastric outlet obstruction to heal any active ulcers

**Prostaglandin Analogues**

- Eg Misoprostol
- Protect the gastroduodenal mucosa by promoting the secretion of bicarbonate & mucus, & by augmenting mucosal blood flow & cell restoration in the gastric mucosa
- Also has antisecretory effect when given in high doses
  - Useful in healing ulcers & preventing ulcer recurrence
- Primarily recommended for prevention of NSAID-induced gastroduodenal ulceration
- Needs frequent dosing & associated w/ more side effects than $H_2$RAs, hence, generally not used for the treatment of PUD
- Major drawback is the incidence of diarrhea & has abortifacient properties, hence, not for use by pregnant women & women of childbearing age

**Sucralfate**

- Has been used to treat PUD & has healing rates same as w/ antacids & $H_2$RAs
- Protects the gastroduodenal mucosa by adhering to the base of the ulcer, adsorbing bile acids, inactivating pepsin, & stimulating bicarbonate & mucus secretion; however, has no effect on gastric acid secretion
- May also be used in preventing duodenal ulcer relapse
- Has an excellent safety profile & is generally well tolerated
- Treatment duration for PUD is 4 wk

## C SURGERY

- Recommended in patients w/ more emergent complications (ie hemorrhage, perforation, gastric outlet obstruction), refractory disease & intractability, or rare causes of ulcer disease (eg gastrinoma, Zollinger-Ellison syndrome)
  - The most common indication for surgery is bleeding
  - Peritonitis is a surgical emergency that needs patient resuscitation, laparotomy & peritoneal toilet, or omental patch placement
- Indications for elective peptic ulcer surgery include resection of ulcers suspected to be malignant, failure to heal despite maximal medical therapy, intolerance or noncompliance w/ medical regimen, & relapse while on maximal medical therapy
- Options for duodenal ulcer may include truncal vagotomy, selective vagotomy, highly selective vagotomy, partial gastrectomy
- Options for gastric ulcer may include partial gastrectomy w/ gastroduodenal or gastrojejunal anastomosis
- Options to relieve chronic obstruction may include vagotomy & pyroplasty, antrectomy, or gastroenterostomy

# Dosage Guidelines

## ANTACIDS[1]

| Drug | Available Strength | Dosage | Remarks |
|---|---|---|---|
| Alexitol sodium (Na polyhydroxy-aluminum monocarbonate-hexitol complex) | 360 mg/tab | 1-2 tab PO ½-1 hr after each meal<br>**Max dose:** 16 tab/24 hr | • May cause constipation, hypophosphatemia & rarely osteomalacia<br>• Al accumulation may occur in renal impairment<br>• Administer other medications 2-3 hr apart to avoid drug interaction<br>• Drug interactions may be caused by increased gastric pH or by drug adsorption in the gut<br>• Large doses can cause intestinal obstruction |
| Aluminium hydroxide [$Al(OH)_3$] | 233 mg/tab | 2-4 tab PO 6-8 hrly | |
| | 360 mg/tab | 1-4 tab PO up to 6 hrly | |
| | 600 mg/tab | 1-2 tab PO 6 hrly | |
| | 320 mg/5 mL oral susp | 5-30 mL PO as required between meals & at bedtime | |
| Aluminium phosphate | 130 mg/sachet | 1 sachet PO 8-12 hrly | |
| Calcium carbonate ($CaCO_3$) | 500 mg/tab | 1-3 tab PO as required<br>**Max dose:** 16 tab/day | • May cause constipation<br>• Administer other medications 2-3 hr apart to avoid drug interactions<br>• Drug interactions may be caused by increased gastric pH or by drug adsorption in the gut |
| Hydrotalcite | 500 mg/tab | 2 tab PO 6-8 hrly | • Hydrated form of Al/Mg basic carbonate |
| | 100 mg/mL oral susp | 10 mL PO 6-8 hrly | |
| Magaldrate | 400 mg/tab | 1-4 tab PO after meals & at bedtime | • Combination of Al & $Mg(OH)_2$ & sulfates |
| | 400 mg/5 mL oral susp | 5-20 mL PO after meals & at bedtime | |
| Magnesium hydroxide [$Mg(OH)_2$] | 311 mg/tab | 2-4 tab PO 1 hr after meals | • May cause diarrhea which is dose-dependent<br>• Hypermagnesemia may occur in patients w/ impaired renal function<br>• Administer other medications 2-3 hr apart to avoid drug interactions<br>• Drug interactions may be caused by increased gastric pH or by drug adsorption in the gut |
| | 400 mg/5 mL oral susp | 5-15 mL PO up to 6 hrly | |
| Magnesium oxide (MgO) | 250 mg/tab | ½-1 tab PO 6-8 hrly | |
| Sodium bicarbonate ($NaHCO_3$) | 325 mg/tab | 2-4 tab PO 8 hrly | • Drug interactions may be caused by increased gastric pH or increased urinary pH which may affect drug elimination |

# Dosage Guidelines

## HISTAMINE-2 RECEPTOR ANTAGONISTS ($H_2$RAs)

| Drug | Dosage | Remarks |
|---|---|---|
| Cimetidine | **Active duodenal ulcer or benign gastric ulcer:** 200 mg PO 8 hrly & 400 mg PO at bedtime *or* 300 mg PO 6 hrly *or* 400 mg PO 12 hrly *or* 600 mg PO 12 hrly *or* 800 mg PO at bedtime<br>**Duration:** 4-8 wk<br>***or***<br>200-300 mg IM/slow IV 4-6 hrly *or* 200-400 mg hrly intermittent IV infusion 4-6 hrly *or* 50-100 mg/hr continuous IV infusion<br>**Max dose:** 2.4 g/day<br>**NSAID-induced ulcer:** 800 mg/day<br>**Maintenance/prevention:** 300-400 mg PO 12 hrly or at bedtime | **Adverse Reactions**<br>• CNS effects (headache, dizziness, somnolence, agitation); GI effects (diarrhea, N/V); Other effects (rashes, myalgia, arthralgia)<br>• Altered LFTs, reversible confusion in the elderly & those w/ renal failure have occasionally occurred<br>• Rarely reported effects: hepatotoxicity, hypersensitivity reactions, hematologic effects (leucopenia, thrombocytopenia, agranulocytosis), CV effects (tachycardia, bradycardia, hypotension), acute pancreatitis<br>• **Cimetidine**: has weak anti-androgenic effects; impotence & gynecomastia have occurred & are usually reversible<br>**Special Instructions**<br>• Intravenous injections should be given slowly; intravenous infusion is preferred (esp for high doses & in patients w/ CV impairment)<br>• Use w/ caution in patients w/ hepatic & renal impairment; dose adjustment recommended<br>• **Cimetidine:** may reduce hepatic metabolism of some drugs through inhibition of cytochrome P450 isoenzymes; closely monitor those on oral anticoagulants, Lidocaine, Phenytoin or Theophylline; dose reduction may be necessary |
| Famotidine | **Active duodenal ulcer or benign gastric ulcer:** 40 mg PO 24 hrly at bedtime or 20 mg PO 12 hrly x 4-8 wk<br>***or*** 20 mg IV/IM 12 hrly<br>**Maintenance/prevention:** 20 mg PO 24 hrly at bedtime | |
| Nizatidine | **Active duodenal ulcer, benign gastric ulcer or NSAID-induced ulcer:** 150 mg PO 12 hrly or 300 mg PO at bedtime for 4-8 wk<br>***or***<br>300 mg continuous IV infusion at a rate of 10 mg/hr *or* 100 mg IV given over 15 min 8 hrly<br>**Max IV dose:** 480 mg/day<br>**Maintenance/prevention:** 150 mg PO 24 hrly | |
| Ranitidine | **Active duodenal ulcer or benign gastric ulcer:** 150 mg PO 12 hrly or 300 mg PO at bedtime x 4-8 wk<br>***or***<br>50 mg IM/slow IV 6-8 hrly *or* 50 mg slow IV then 0.125-0.25 mg/kg/hr continuous IV infusion<br>**NSAID-induced ulcer:** 150 mg PO 12 hrly or 300 mg at bedtime for 8-12 wk<br>**Maintenance/prevention:** 150 mg PO at bedtime | |
| Roxatidine | **Active duodenal ulcer or benign gastric ulcer:** 75 mg PO 12 hrly or 150 mg PO at bedtime | |

# Dosage Guidelines

## PROTON PUMP INHIBITORS (PPIs)

| Drug | Dosage | Remarks |
|---|---|---|
| Esomeprazole | 20-40 mg PO 24 hrly<br>**NSAID-induced ulcer:** 20 mg PO/slow IV 24 hrly x 4-8 wk<br>**Prophylaxis for NSAID-associated ulcer:** 20-40 mg PO 24 hrly<br>***or***<br>20 mg IV 24 hrly<br>**Maintenance of hemostasis & prevention of ulcer rebleeding:** 80 mg IV infusion for 30 min, followed by 8 mg/hr continuous IV given over 3 days, followed by 40 mg PO once daily for 4 wk | **Adverse Reactions**<br>• Generally well-tolerated; most commonly reported: headache, diarrhea, rash<br>• Less common: dizziness, fatigue, GI effects (constipation, flatulence, abdominal pain, N/V, dry mouth); Dermatologic effects (pruritus, urticaria); Musculoskeletal effects (arthralgia, myalgia); Other effects (dizziness, fatigue, insomnia)<br>• Hypersensitivity reactions, elevated liver enzymes, & isolated cases of photosensitivity & hepatotoxicity have been reported<br>**Special Instructions**<br>• Use w/ caution in patients w/ hepatic impairment; dose adjustment recommended<br>• Concomitant use w/ Atazanavir is not recommended (PPIs reduce exposure to Atazanavir) |
| Lansoprazole | **Active duodenal ulcer:** 30 mg PO 24 hrly x 4-8 wk<br>**Benign gastric ulcer:** 30 mg PO 24 hrly x 8 wk<br>**Maintenance:** 15 mg PO 24 hrly<br>**NSAID-induced ulcer:** 15-30 mg PO 24 hrly x 4-8 wk<br>**Prophylaxis for NSAID-associated ulcer:** 15-30 mg PO 24 hrly | |
| Omeprazole | **Active duodenal ulcer:** 20 mg PO 24 hrly x 2-4 wk may be increased to 40 mg in severe cases<br>**Active duodenal ulcer:** 20 mg PO 24 hrly x 4-8 wk may be increased to 40 mg in severe cases<br>***or***<br>40 mg slow IV inj/infusion 24 hrly for 5-7 days<br>**Maintenance/prevention:** 10-20 mg PO 24 hrly<br>**NSAID-induced ulcer:** 20 mg PO 24 hrly x 4-8 wk<br>**Prophylaxis for NSAID-associated ulcer:** 20 mg PO 24 hrly | |
| Pantoprazole | **Active duodenal ulcer:** 40 mg PO 24 hrly x 2-4 wk<br>**Benign gastric ulcer:** 40 mg PO 24 hrly x 4-8 wk<br>***or***<br>40 mg/day for 1 wk<br>**Max duration:** 8 wk<br>**NSAID-induced ulcer:** 20 mg PO 24 hrly<br>**Prophylaxis for NSAID-associated ulcer:** 20 mg PO 24 hrly | |
| Rabeprazole | **Active duodenal ulcer:** 10-20 mg PO 24 hrly in the morning x 4-8 wk<br>**Benign gastric ulcer:** 10-20 mg PO 24 hrly in the morning x 6-12 wk | |

# Dosage Guidelines

## PROSTAGLANDIN

| Drug | Dosage | Remarks |
|---|---|---|
| Misoprostol | **Active duodenal ulcer, benign gastric ulcer, NSAID-induced ulcer:** 800 mcg/day PO divided 6-12 hrly for 4-8 wk<br>**Prophylaxis for NSAID-associated ulcer:** 100-200 mcg PO 6-12 hrly | **Adverse Reactions**<br>• Most common: diarrhea<br>• GI effects (abdominal pain, dyspepsia, flatulence, N/V); Gynecologic effects (increased uterine contractility, abnormal vaginal bleeding); Other effects (rash, headache, dizziness, rarely hypotension)<br>**Special Instructions**<br>• Should not be used in women of childbearing potential unless able to comply w/ effective contraception<br>• Use w/ caution in elderly, in patients w/ cardiovascular disease, renal impairment, inflammatory bowel disease |

## OTHERS

| Drug | Dosage | Remarks |
|---|---|---|
| Cetraxate | 200-300 mg PO 6-8 hrly | **Adverse Reactions**<br>• Thirst, GI disturbances, exanthema, hypersensitivity<br>**Special Instructions**<br>• Use w/ caution in patients w/ thrombosis, consumption coagulopathy |
| Pirenzepine | 25-50 mg tid for 4-6 wk<br>***or***<br>10 mg slow IV/IM 12 hrly | **Adverse Reactions**<br>• Antimuscarinic effects such as dry mouth or blurring of vision<br>**Special Instructions**<br>• Use w/ caution in patients w/ renal impairment esp those w/ end stage renal failure |
| **Bismuth Preparations** | | |
| Bismuth salicylate (Bismuth subsalicylate) | 262-524 mg PO 1/2-1 hrly<br>**Max dose:** 8 doses/day | **Adverse Reactions**<br>• Not common if used for limited periods<br>• Excessive dose or long-term use: CNS effect (reversible encephalopathy); GI effects (N/V, stomatitis, darkening of feces & tongue); Other effects (bone & joint toxicity, skin reactions, renal failure, liver damage)<br>• Bismuth salicylate may have same side effects like Aspirin: GI effects (N/V, dyspepsia, GI ulceration); Hematologic effects or hypersensitivity reactions<br>**Special Instructions**<br>• Contraindicated in patients w/ moderate-severe renal impairment; Salicylate formulation should be avoided in patients w/ history of GI bleeding or coagulopathy<br>• Bismuth salicylate should be used w/ caution in patients taking Aspirin |
| Bismuth subcitrate (Colloidal bismuth subcitrate) | 120-300 mg PO qid<br>*or*<br>240-600 mg PO bid x at least 1 mth followed by interval of 2 mth treatment free | |

# Peptic Ulcer Disease (1 of 11)