

- **Lifestyle modification: 1st line**

- Head of bed elevation

- Avoiding smoking, heavy alcohol consumption, large evening meals, nighttime snacks, and high dietary fat intake.

- **Weight loss is encouraged in overweight GERD patients.**

- **All patients with GERD should avoid NSAIDs** because of their role in disrupting physiologic mucosal protection mechanisms.

- **Drug therapy:-**

1. Antiacids → work by chemically neutralizing the acid without interacting with any receptors or enzymes (**given on occasional symptoms**)
2. H2 histamine antagonists/ blockers (**given daily**)
3. Proton pump inhibitors (**given daily**)

## **Antacids:-**

Antiacids are **weak bases**.

**Neutralize gastric acidity** by :-

- 1- **Reacting with gastric acids** to form salts and water
- 2- **Diminishing pepsin activity** as pepsin is inactive at PH above 4

Antiacids vary in their chemical composition , acid neutralizing capacity, **sodium content**(beware of renal failure patients!), and Palatabilityالمذاق

Antacids			
Aluminum hydroxide:	Magnesium hydroxide:	Calcium carbonate (Tums):	• Sodium bicarbonate:.
<ul style="list-style-type: none"> <li>• Useful in hyperphosphatemia</li> </ul> <p>It binds to phosphate in the gastrointestinal (GI) tract and subsequently prevent the absorption of phosphate.</p> <ul style="list-style-type: none"> <li>• Tend to cause constipation.</li> </ul> <p><u>So if a patient has diarrhea, give aluminum hydroxide</u></p>	<ul style="list-style-type: none"> <li>• Tend to cause diarrhea.</li> <li>• Aluminum hydroxide and magnesium hydroxide are usually combined to minimize the impact upon bowel function.</li> </ul>	<ul style="list-style-type: none"> <li>• Excessive dose can lead to hypercalcemia.</li> </ul>	<ul style="list-style-type: none"> <li>• Systemic absorption can cause transit metabolic alkalosis and produce significant sodium load (specially in patients with heart failure, hypertension and renal insufficiency) .</li> <li>Not recommended</li> </ul>

## Antacids duration of action

- Antacids afford fast symptoms relief.
  - Have short duration of action.
  - Duration of action of an antacid is related primarily to the length of time the medication remains in the stomach:
- In the fasting state, the duration of action is relatively short and can be prolonged by giving an antacid after food.

## Contraindications and precautions

- Absorption of cations (Mg, Al, Ca) from antacids is usually not a problem in patients with normal renal function, however, accumulation and adverse effect may occur in patients with renal failure.
- Chronic administration of calcium carbonate containing antacids should be avoided because of hypercalcemia.

## Drug interaction

- All antacid may affect the absorption of other medications by:
  - 1- Binding the drug → reducing its absorption
  - 2- Increasing intragastric PH so that the drug solubility is altered. So antacid shouldn't be given within 2 hours of doses of tetracycline, fluoroquinolone, itraconazole and iron.

## GERD during pregnancy

Mild to moderate: antacid or H2 receptor antagonist.

Severe : PPIs

## Peptic ulcer

Peptic ulcers usually occur due to <b>imbalance</b> in <b>aggressive factors like HCl</b> and <b>defensive factors like mucus</b> in stomach lining	
<b>Aggressive factors</b>	<b>Defensive factors</b>
<b>Endogenous:</b> 1. Hcl. 2. Pepsin. 3. Bile.	<b>Mucus.</b> <b>Na HCO<sub>3</sub>.</b> <b>Integrity of gastric mucosa:</b> tight junctions between cells prevent back diffusion of Hcl. <b>Prostaglandins :</b> decreases with age, with increased incidence of PU.
<b>Exogenous:</b> 1. Smoking 2. Stress	<b>NSAID Inhibits prostaglandin via inhibiting Cox-1 and Cox-2</b>

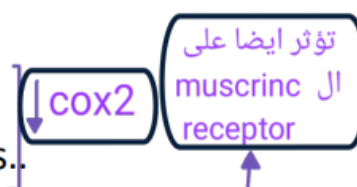
Other causes of ulcer(they might come in a case):-

Idiopathic, hypersecretion of gastric acids (Zollinger-Ellison syndrome), viral (CMV), vascular insufficiency (cocaine), radiation, Chemotherapy, infiltrating disease (Crohn's disease).

أدوية نحاول نخفف منها عند مرضى ال ulcer

## Drugs increasing Hcl secretion

- 1) **NSAIDs** : by decrease prostaglandins.
- 2) **Corticosteroids** : by decrease prostaglandins..
- 3) **Nicotine** (smoker's ulcer): by stimulation of nicotinic receptors.
- 4) **Cholinomimetics** : by muscarinic M3 receptor stimulation.
- 5) **Alpha methyl dopa**: by **parasympathetic predominance**.
- 6) **Gastrin**: by stimulation of gastrin receptors.
- 7) **Histamine**: by stimulation of H<sub>2</sub> receptors.
- 8) **Calcium**: increases gastrin → **long term**
- 9) **Caffeine**: by increase cAMP.
- 10) **Alcohol**.



### Risk Factors Associated with (NSAID)–Induced Ulcers

- Age >65
- Previous peptic ulcer
- Previous ulcer-related upper GI complication
- High-dose NSAIDs
- Multiple NSAID use
- Selection of NSAID (selective COX-2 inhibitors are less likely to cause ulcers)
- NSAID-related dyspepsia
- Aspirin (including cardioprotective dosages)
- Concomitant use of :

NSAID plus low-dose aspirin

Oral bisphosphonates(e.g., alendronate)

Corticosteroids Anticoagulant or coagulopathy

Antiplatelet drugs (e.g., clopidogrel)

Selective serotonin reuptake inhibitor

## Diagnosis of peptic ulcer

**Epigastric pain and point tenderness, rhythmicity (relation to meals, nocturnal) & periodicity (symptoms for weeks or months).**

**DU: fasting or nocturnal, relieved by food.**

**GU: increased by food.**

For H pylori ulcer diagnosis: **urea breath test or fecal antigen test.**

**Stop antimicrobials 4 weeks, PPIs 2 weeks and H2 antagonists 24 hours before.** Endoscopy with biopsy for H pylori and to exclude malignancy. Cancer in GU is > DU.