### Acute and chronic gastritis

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### Introduction

- Gastritis is an inflammation of the gastric mucosa.
- it is classified as either acute or chronic.
- Incidence high in 5<sup>th</sup> 6<sup>th</sup> decades of life.
- M> F.
- Increases with smoking and alcohol consumption.

### **Acute gastritis**

- Acute gastritis is usually short duration unless the gastric mucosa showed extensive damage.
- Etiology:
- Ingestion of a corrosive, erosive substance.
- Drugs: aspirin, NSAID, chemotherapy, steroids.
- Systemic bacterial or viral infection.
- Acute alcohol and food poisoning.
- Ischemia, shock & uremia.

## pathophysiology

The mucosal lining of the stomach normally protects it from the action of gastric acid. This mucosal barrier is composed of prostaglandins.

Due to any cause

This barrier is penetrated

Hydrochloric acid comes into contact with the mucosa

Injury to small vessels

Edema, hemorrhage, and possible ulcer formation

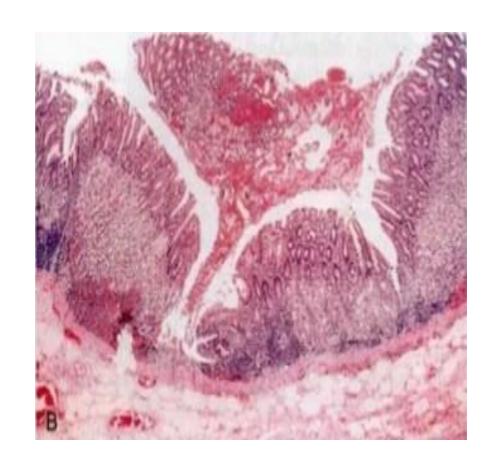
# Clinical picture

- Abdominal pain and tenderness,
- Cramping.
- Belching.
- Reflux.
- Nausea and vomiting.
- Hematemsis.
- Diarrhea.

### Diagnosis

- History of food intake, medications, and any disorder related to gastritis.
- Physical examination, rectal examination.
- <u>Investigations</u>:
- H.pylori test.
- Stool routine examination and for occult blood.
- Upper GIT endoscopy and histological examination by biopsy of the sample.

- The gastric mucosa is edematous, with neutrophilic infiltrations.
- Mucosal erosions, ulceration and hemorrhage.



### **Treatment**

- Stop consumption of any factor that precipitate inflammation.
- Antiemetic drugs. Meticulopromide, domperidone.
- Antiacids; cimetidine, ranitidine,
- Proton pump inhibitors.
- Treat the under lying cause of gastritis.

### Dietary therapy

- Initially foods and fluids are with held untill N& V subside.
- Avoid spicy food, caffeine and large heavy meals. Then can slowly return to a normal diet if the symptoms subsides

### **Chronic gastritis**

#### Superficial gastritis:

Reddened, edematous mucosa with small erosions and hemorrhage.

#### Atrophic gastritis:

Decreased number of parietal and chief cells, in association with gastric ulcer and gastric cancer. presents in pernicious anemia.

#### Hypertrophic gastritis:

Thickened and nodular mucosa. Hemorrhage occure frequently.

### Etiology

- Peptic ulcer disease.
- After gastrojejunostomy.
- H. pylori infection (chronic. Atrophic non atrophic gastritis).
- Age: risk of chronic gastritis increases with age.
- Radiation injury
- Chronic bile reflux

## Pathophysiology

The stomach lining first becomes thickened and erythematous and then becomes thin and atrophic.

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Continued deterioration and atrophy



Loss of function of the parietal cells



Acid secretion decreases

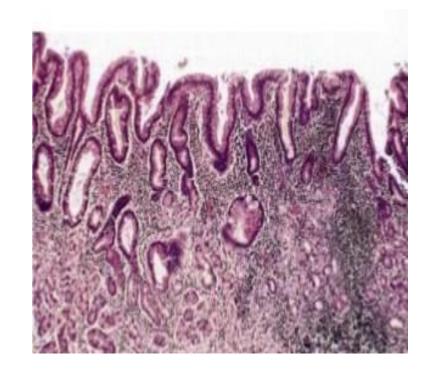


Inability to absorb vitamin B12



Development of pernicious anemia

- Infiltration of lymphocytes &/ or plasma cells in the lamina propria.
- Partial replacement of the gastric mucosal epithelium by intestinal metaplasia



## Clinical picture

- Anorexia
- Feeling of fullness
- Dyspepsia
- Belching
- Vague epigastric pain
- N&V
- Intolerance to spicy and fatty food.

## complication

- Bleeding
- Pernicious anemia
- gastric cancer

### Management

- Small frequent meals
- PPI
- H.pylori eradication
- Intramuscula injections of vit B 12 monthly if pernicious anemia develops.
- Dietary management
- Anxiety related treatment

# Thank you