# Gastroenterology cases GP Symposium 2024

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Gastroenterologist and Hepatologist

# Content

Case 1

Upper GI symptoms

Case 2

Lower GI symptoms

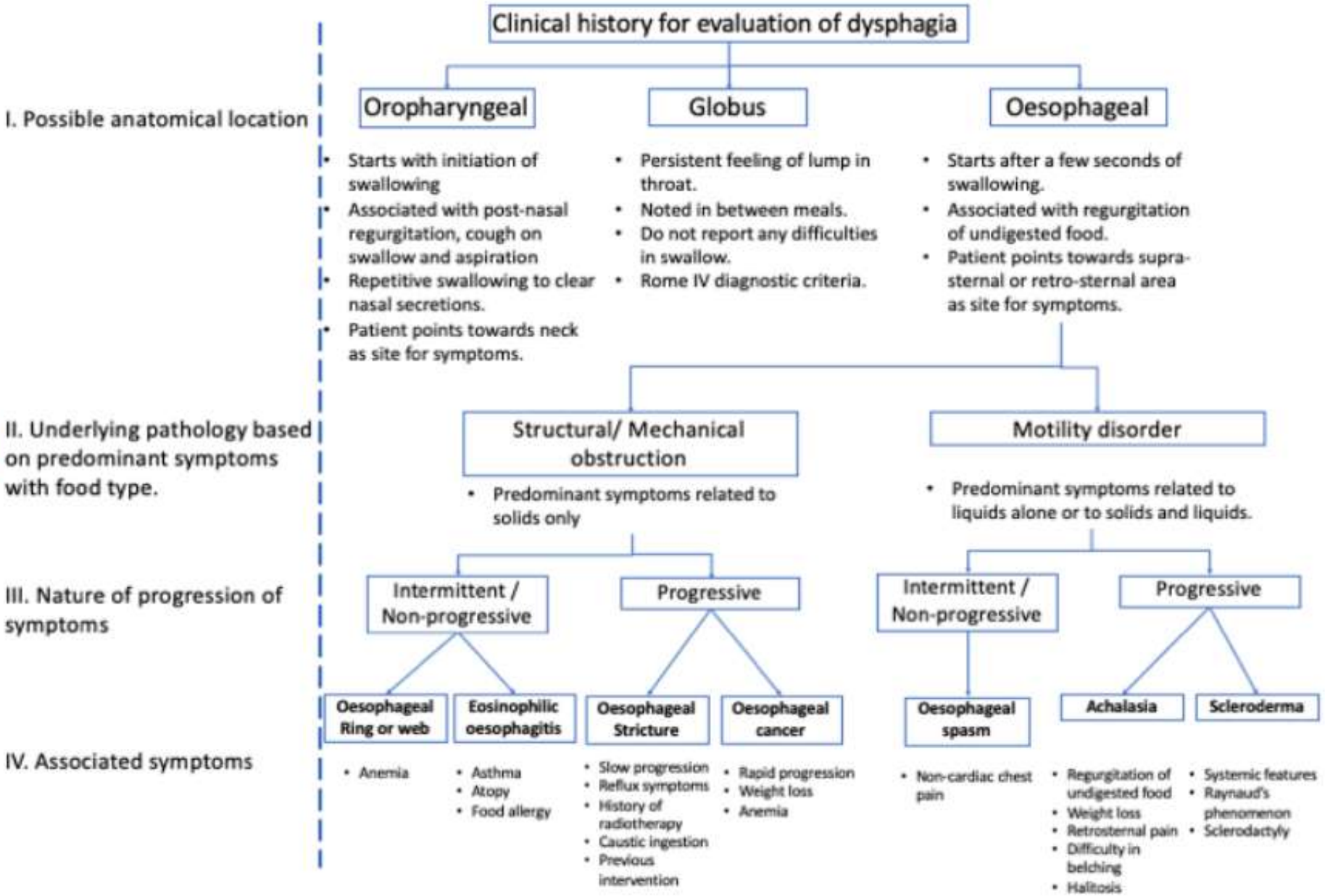
# Case 1

- 34 years old man presented with intermittent dyspepsia and dysphagia 2 years.
- He noted that occasionally he feels food get stuck in oesophagus but after a few minutes these symptoms resolve after glass of water
- Never had a presentation with food bolus
- No issues with swallowing fluids
- Gets reflux symptoms and takes Omeprazole 20mg every morning for last 9 months and symptoms doesn't really improve.
- No LOA, no LOW, no GI bleeding symptoms, no nausea or vomiting

- PMHx: Nil
- FHx: Father has MI and diabetes, Mother has history of asthma
- Social Hx: Works as an electrician, does not smoke, social ETOH
- Physical Exam: Unremarkable

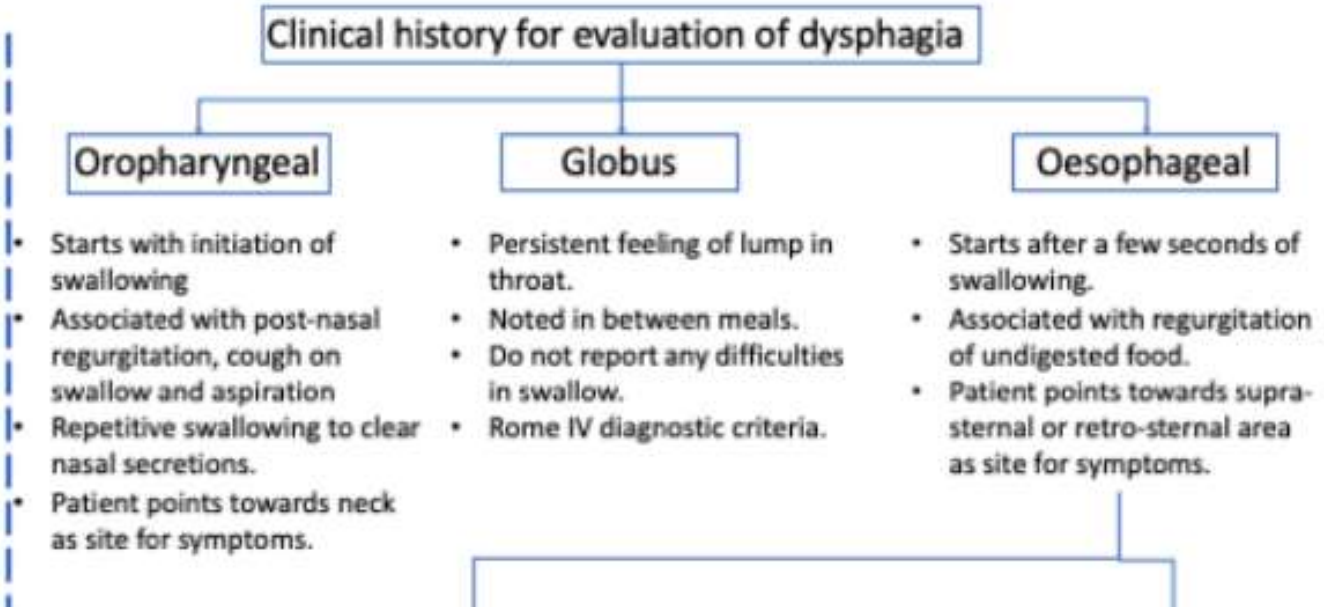
# Laboratory

- Hb: 142 g/L (130-175)
- WCC: 7.0 x10e9/L (4.0-11)
- Plt: 266x10e9/L (150-400)
- Na: 142 mmol/L (135-145)
- K: 4.0 mmol/L (3.5-5.2)
- Creatinine: 95umol(60-105)
- LFT- normal
- CRP-3




Nigam GB, Vasant DH, Dhar A
Curriculum review : investigation and management of dysphagia
*Frontline Gastroenterology* 2022;**13:**254-261.

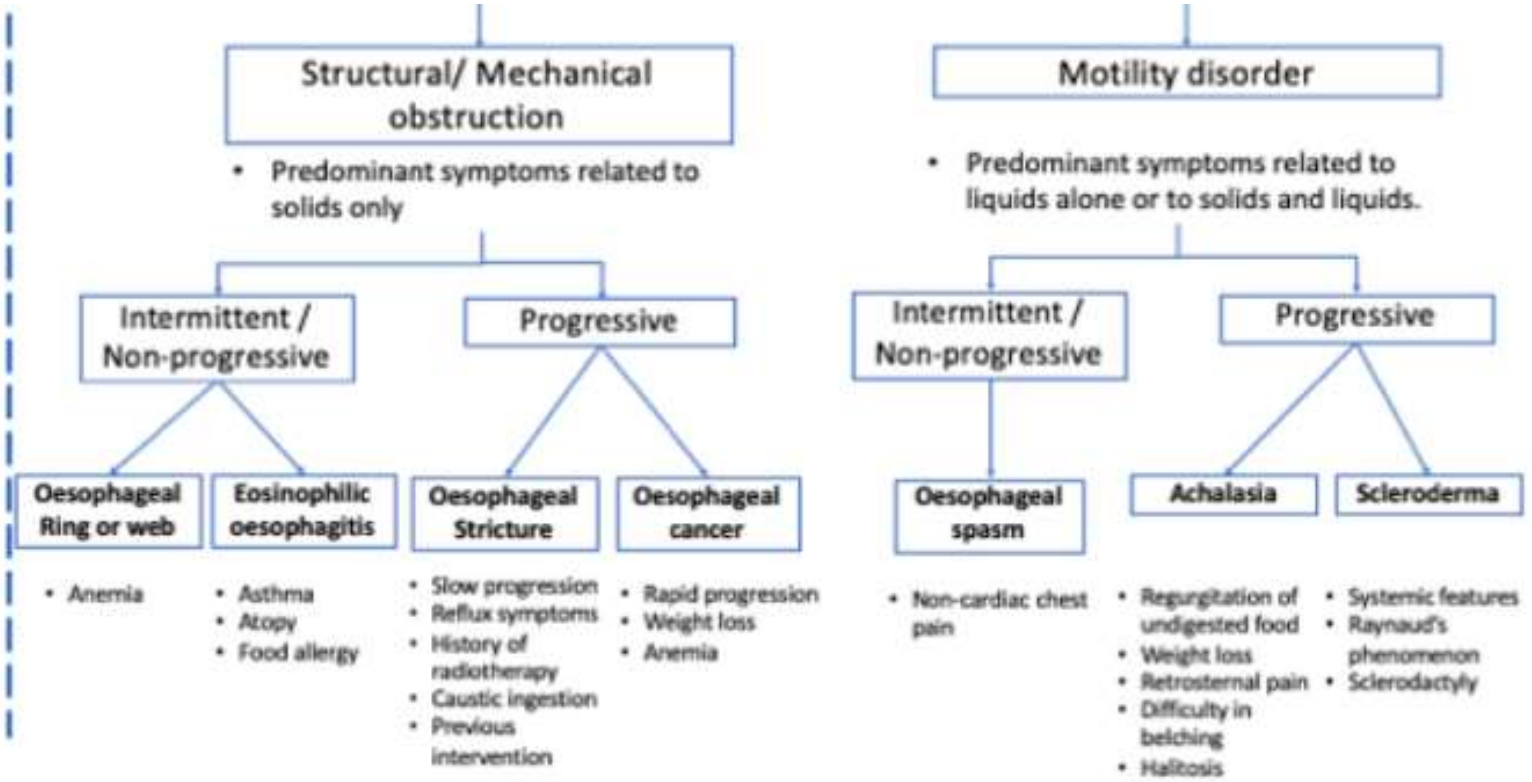
I. Possible anatomical location



II. Underlying pathology based on predominant symptoms with food type.

III. Nature of progression of symptoms

IV. Associated symptoms

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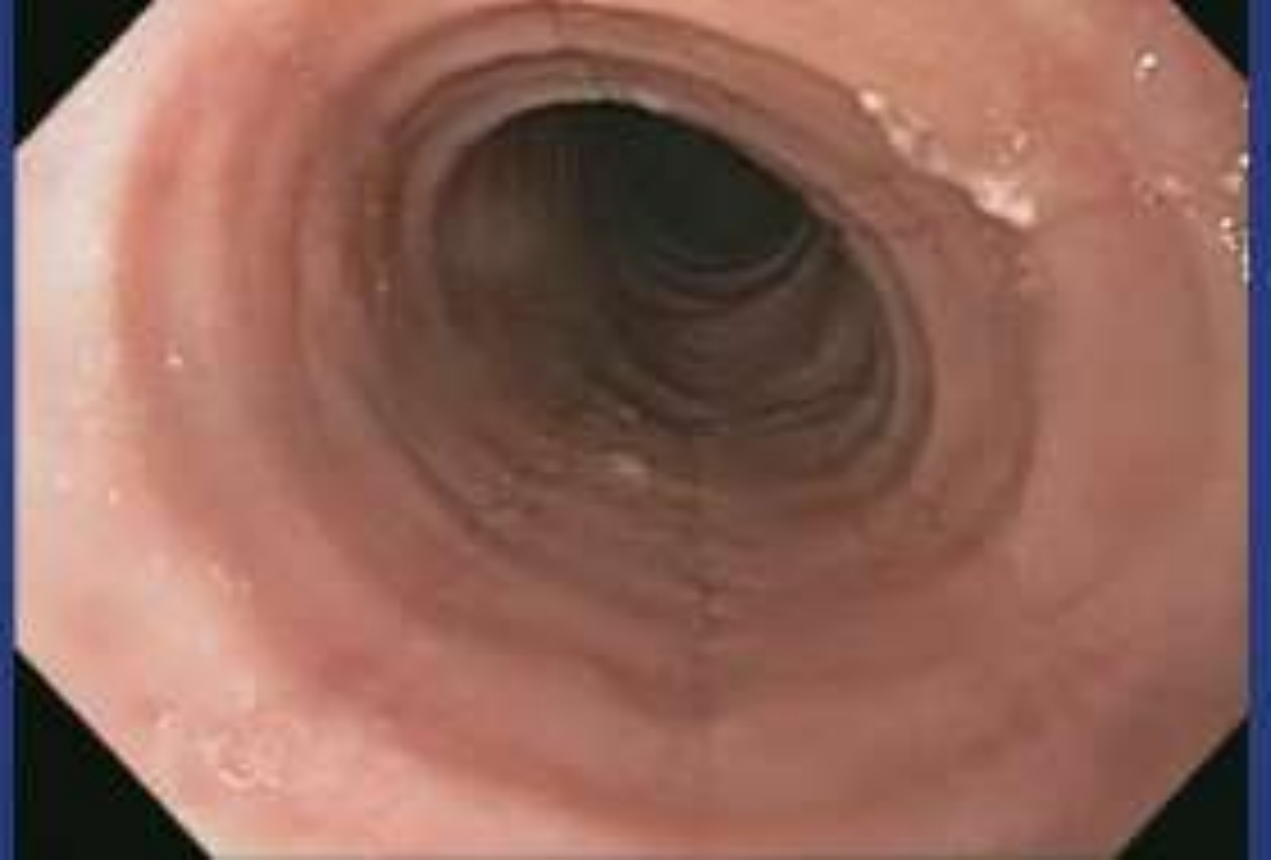
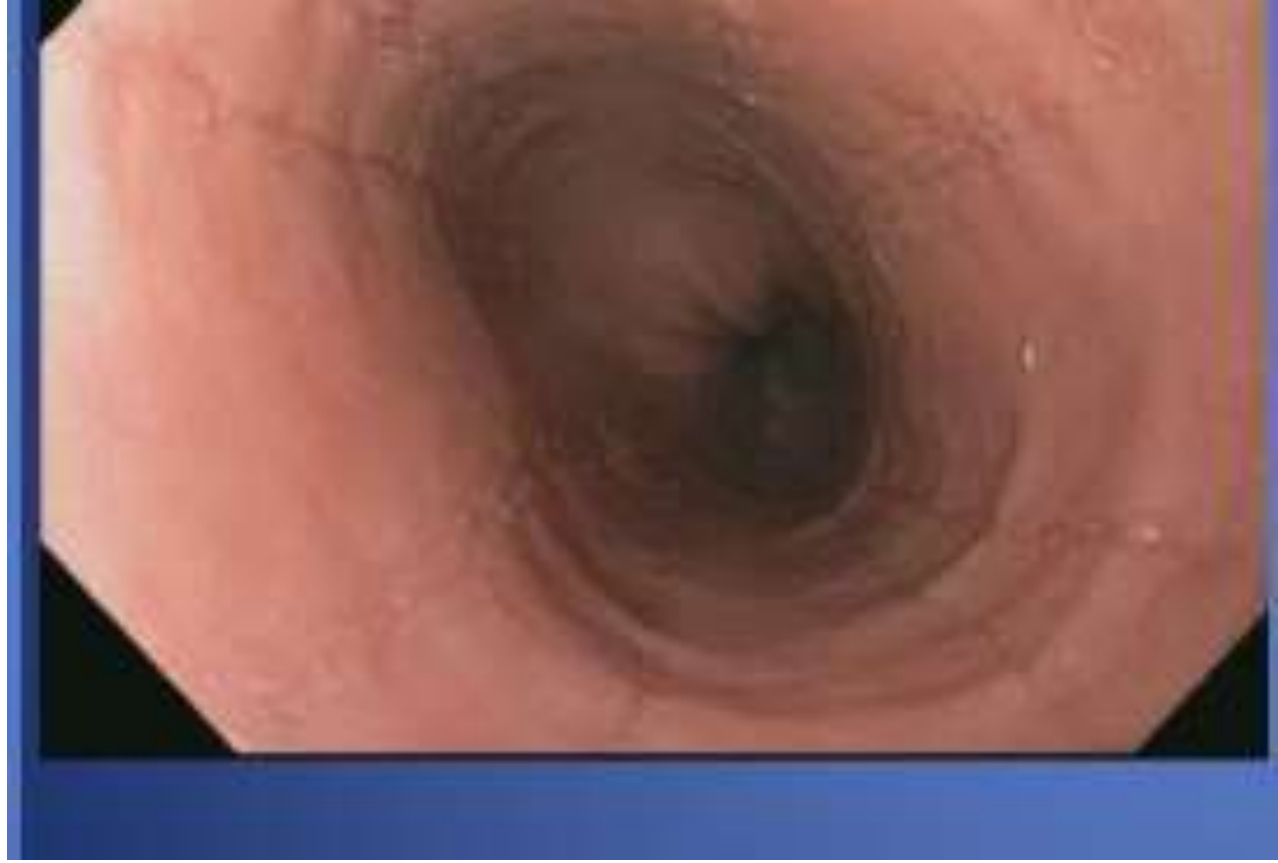




# Referred to Gastroenterology for endoscopy

# Histology

- Eosinophilic Esophagitis

# Eosinophilic Esophagitis

- Chronic immune or antigen-mediated process
- Presents with various esophageal dysfunction
- Etiology of EoE is unknown- result of the interactions of environmental, genetic, and host immune factors.
- Correlation between atopy and EoE

# Epidemiology

- The first cases were described in the late 1970s and it was defined as an entity in the early 1990s
- EoE is common in both pediatric and adult populations.
- Based on many population studies, the reported incidence of EoE is between 0.1/10,000 to 1.2/10,000 worldwide.
- EoE can occur in all age groups; however, it is most common in men during their 20s and 30s, and the mean age of diagnosis is 34

# Diagnosis

- Clinical
- Endoscopic
- Histopathology

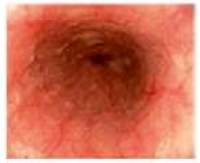
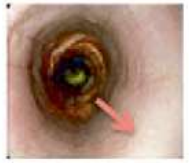
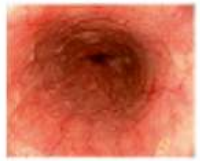
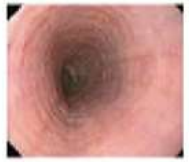
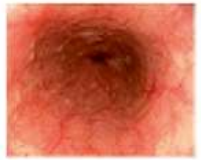
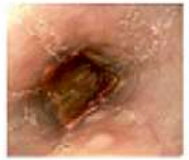
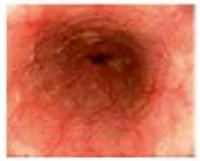
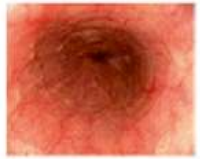
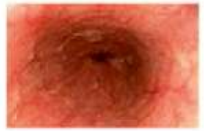
# Clinical Features

- Overlapping symptoms with GERD
- Most common manifestation is dysphagia to solid food
- Pediatric patients can present with nausea, vomiting or food intolerance
- A history of various atopic conditions such as asthma, atopic dermatitis, seasonal allergy, food allergy, allergic rhinitis, and eczema may be present as well.

# Endoscopy

- Upper endoscopy with esophageal biopsy also should be done on patients with a presumed diagnosis of GERD who are resistant to optimal proton pump inhibitor (PPI) dose (20 to 40 mg orally twice daily) and duration (8 to 12 weeks)

## ENDOSCOPIC CLASSIFICATION OF EOSINOPHILIC ESOPHAGITIS

| MAJOR ASPECTS | Grade 0 | Grade 1 | Grade 2 | Grade 3 |
|---|---|---|---|---|
| **Edema** (decreased vascular pattern)<br>Grade 0: absent<br>Grade 1: present |  |  | | |
| **Fixed rings** (trachealization)<br>Grade 0: absent<br>Grade 1: mild (ridges)<br>Grade 2: moderate (defined)<br>Grade 3: severe (the endoscope does not pass) |  |  |  |  |
| **Exudates** (whitish plaques or dots)<br>Grade 0: absent<br>Grade 1: mild (<10% surface)<br>Grade 2: severe (>10% surface) |  |  |  | |
| **Longitudinal furrows**<br>Grade 0: absent<br>Grade 1: present |  |  | | |
| **Stenosis**<br>Grade 0: absent<br>Grade 1: present |  |  | | |
| **MINOR ASPECT** | | | | |
| **Crepe paper mucosa** (fragility/laceration)<br>Grade 0: absent<br>Grade 1: present |  |  | | |

*Modified from: Hirano I, Moy N, Heckman MG, et al. Endoscopic assessment of the Oesophageal features of eosinophilic esophagitis: validation of a novel classification and grading system. Gut. 2013; 62: 489-95.*

# Histopathology

- **The pathological diagnosis of EoE is made when eosinophils are present greater than or equal to 15 per high power field (HPF)**

**Table II. Causes of tissue eosinophilia[15]**

- Inflammatory bowel disease
- Hypereosinophilic syndrome
- Celiac disease
- Drug reactions
- Gastroesophageal reflux*
- Infections (herpes, candida, parasitosis)
- Certain neoplasms
- Vasculitis
- Food protein-induced enterocolitis

**Gastroesophageal reflux disease can induce eosinophilic infiltration, but generally below 5 eos/high-power field (HPF).*

# Treatment and management

**Dietary Treatment**

**Pharmacological Treatment**

**Endoscopic Management**

# Dietary Management

- Patients with a history of atopy to food generally respond well to dietary therapy.
- Six food elimination diet (SFED) - cow's milk, wheat, peanut/tree nut, egg, soy, and seafood/shellfish
- Elemental diet

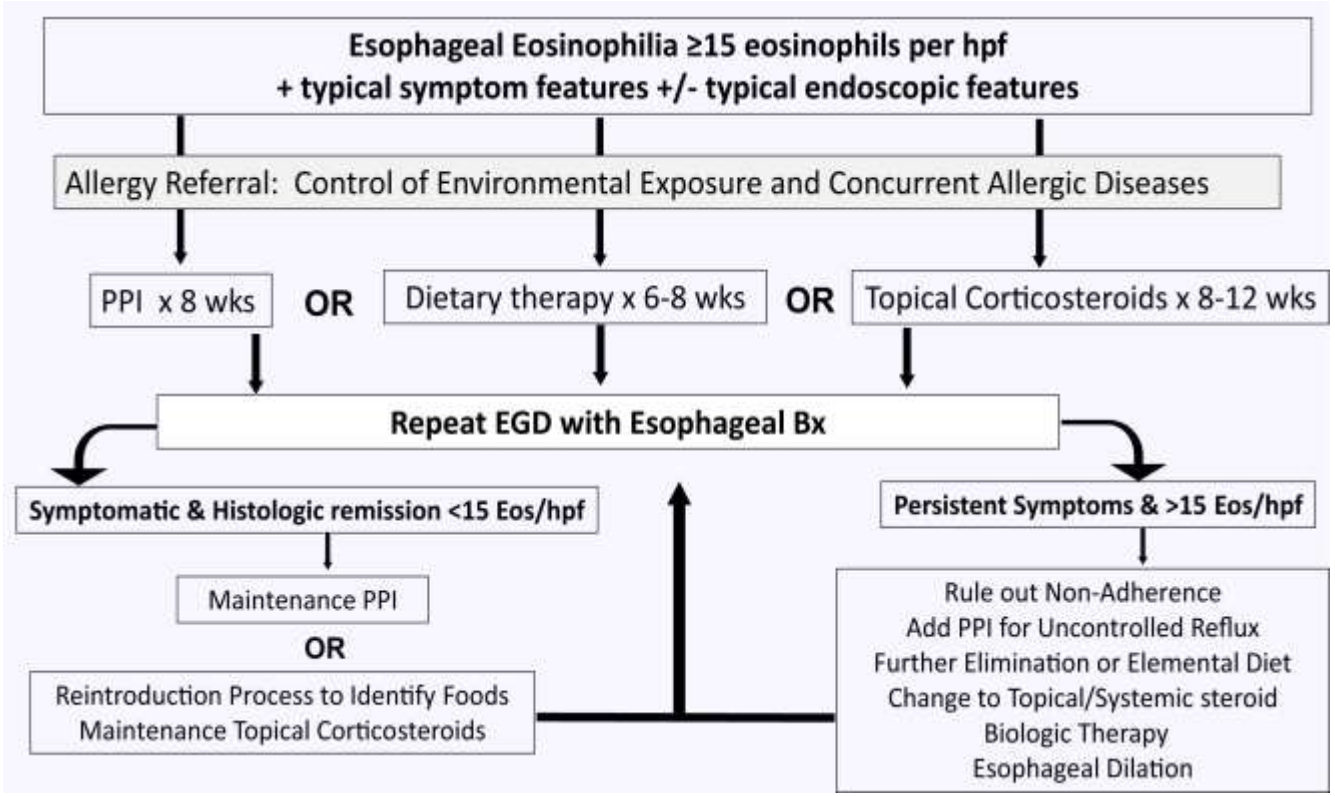
# Pharmacological Treatment

- PPI 20 mg to 40 mg oral daily or twice daily
- If no eosinophils present in repeat biopsy, the diagnosis is either acid mediated GERD with eosinophilia or non GERD PPI responsive EoE with unknown mechanism
- Fluticasone MDI 250mcg per day (Swallowed- 2 puffs BD)
- Oral steroids (2mg/kg)
- Dupilumab

# Endoscopic Treatment

- Food Impaction
- Dilatation

**During the natural course of EoE, progression from an inflammatory to a fibrostenotic phenotype occurs. With each additional year of undiagnosed EoE the risk of stricture presence increases with 9%.**




*Nirmala P. Gonsalves, MD, Seema S. Aceves, MD, PhD Journal of Allergy and Clinical Immunology* Volume 145 Issue 1 Pages 1-7 (January 2020)

# Case 2

- 60 year old lady presented with 1 year history of chronic watery diarrhea
- She describe it as watery and goes around 5 times a day and fecal urgency
- Significant bloating
- No blood in her stools
- No Abdominal pain, no fevers, no nausea, no vomiting
- Denies any weight loss and no oversea travels
- This has been very stressful for her and have not gone out for any activity as fear of needing to go to the toilet

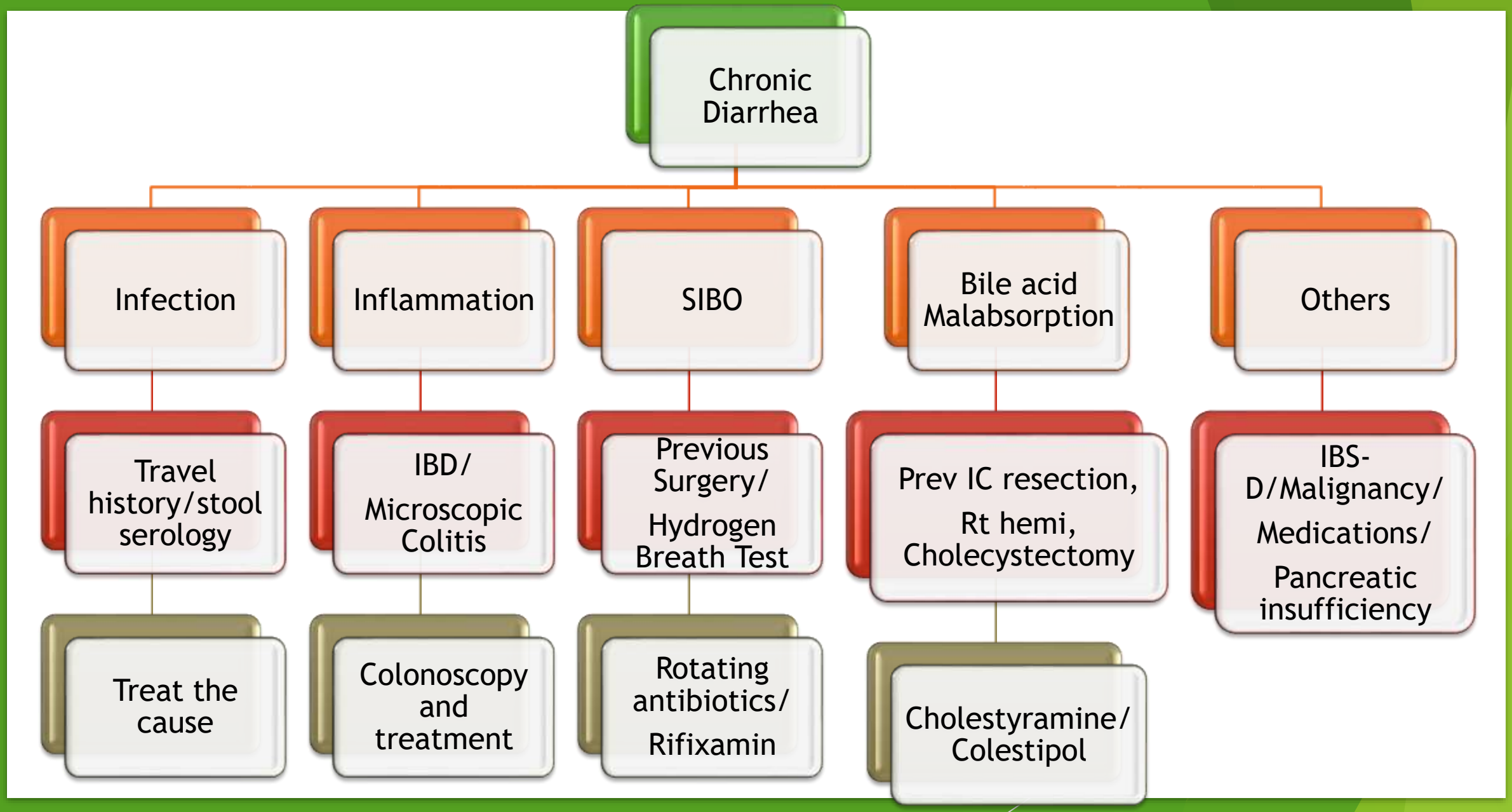
- PMHx: Hypertension, Dyslipidemia
- Past Surgical Hx: Cholecystectomy 8years ago
- Medications: Candesartan, Atorvastatin
- Social Hx: works as lecturer in university, Never smoke and minimal alcohol

- Physical Exam – vitals normal
- BMI 30
- Abdomen: Soft non tender no peritonism

# Labs

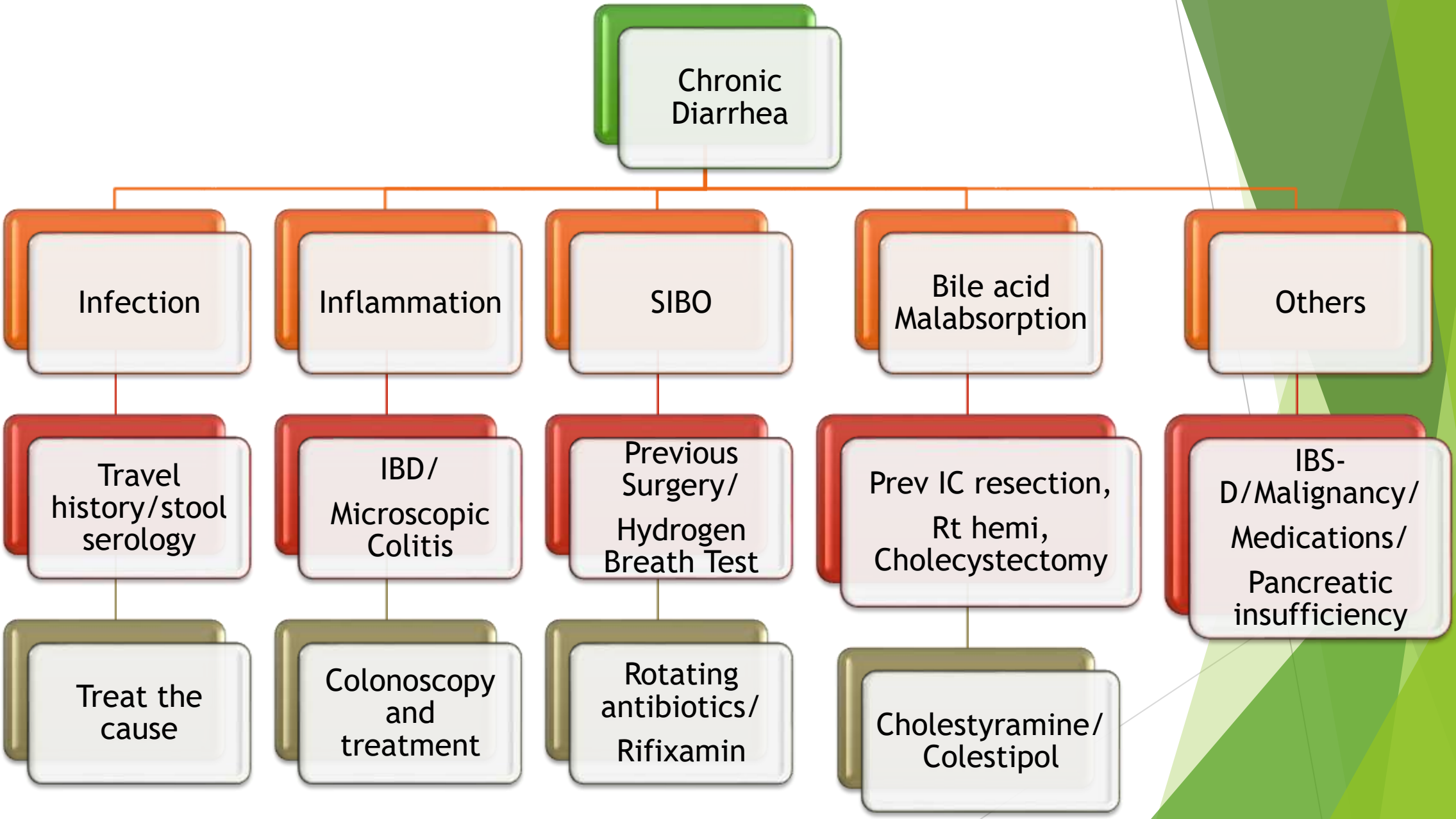
- Full blood count was normal
- LFT mildly Cholestatic – Bil normal, ALP 140 (40-110) GGT 100 (<60) otherwise normal
- CRP 3
- Thyroid function, Celiac screen and extended electrolytes were all normal
- Fecal sample for infection screen including Giardia, Amoeba, Campylobacter was negative
- Fecal calprotectin – <50
- Fecal Elastase and Fecal Steatocrit were normal

# What next?


Chronic Diarrhea
Infection
Travel history/stool serology
Treat the cause
Inflammation
IBD/ Microscopic Colitis
Colonoscopy and treatment
SIBO
Previous Surgery/ Hydrogen Breath Test
Rotating antibiotics/ Rifixamin
Bile acid Malabsorption
Prev IC resection, Rt hemi, Cholecystectomy
Cholestyramine/ Colestipol
Others
IBS-D/Malignancy/ Medications/ Pancreatic insufficiency

# Colonoscopy

- Normal mucosa on endoscopy
- No inflammation noted
- 3 small sessile polyps all – TA LGD
- Colonic biopsies performed- histology was normal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# What should we do next?

- Trial of Cholestyramine

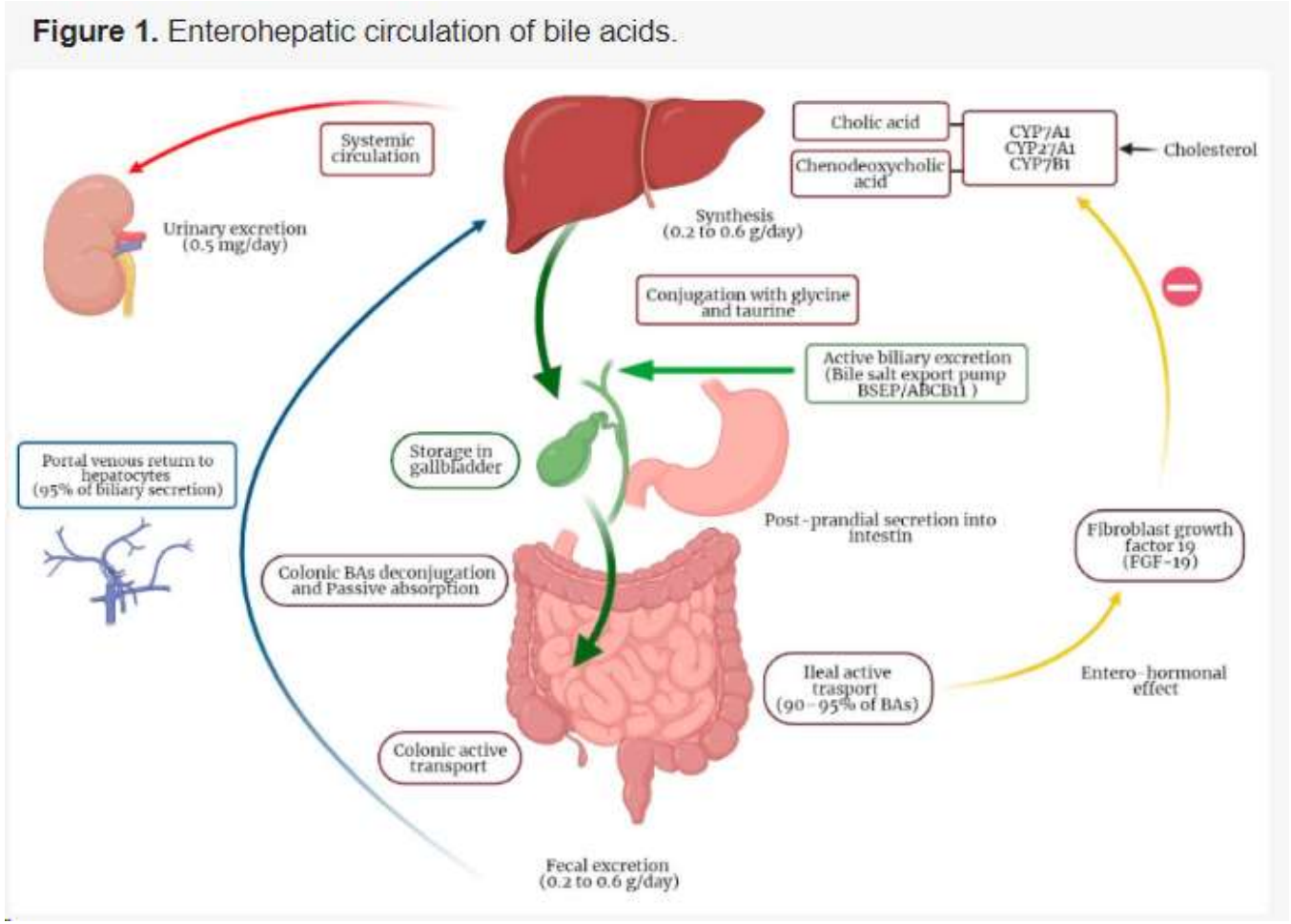
- Significant improvement in symptoms

# Bile Acid Diarrhea (BAD)

# Introduction

- Bile Acid – detergent molecules synthesized in the liver and are responsible for solubilization of fatty acids and monoglycerides facilitating digestion and lipid absorption in the small intestine
- Bile is stored in the gallbladder during fasting; when food reaches duodenum, cholecystokinin is released and stimulates bile secretion into the small intestine.
- In normal conditions, about 90–95% of BA is reabsorbed in the terminal ileum, while the unabsorbed amount of BA reaches the large intestine

**Figure 1.** Enterohepatic circulation of bile acids.



# BAD Classification

- Type 1: Ileal dysfunction and impaired reabsorption, e.g., Crohn's disease
- Type 2: Primary, or idiopathic, BAD produces a similar picture of increased fecal BAs, watery diarrhea, and response to BA sequestrants in the absence of ileal or other obvious gastrointestinal disease
- Type 3: Other gastrointestinal disorders which affect absorption, such as small intestinal bacterial overgrowth, celiac disease, or chronic pancreatitis
- Type 4: BAD may result from excessive hepatic BA synthesis; for example, the oral hypoglycemic drug, metformin, is associated with increased hepatic BA synthesis

# Clinical Manifestation

- Chronic watery diarrhea (80%)
- Fecal Urgency (85%)
- Bloating
- Abdominal discomfort
- Fecal incontinence
- Symptoms impact daily habits and social life

# Diagnostic Test

- Selenium HomotauroCholic Acid Test (75SeHCAT)
- Fasting Serum 7α-hydroxy-4-cholesten-3-one (C4)
- Fecal BA test

# Treatment

- Dietary modification – low fat diet
- Bile Acid *Sequestrants*

- Cholestyramine, colestipol and colesevelam

# Bile Acid Sequestrants

- Poorly tolerated
- Common gastrointestinal side effects such as constipation, abdominal pain, bloating, fullness, nausea and flatulence
- Could interfere with the intestinal absorption of drugs -administer other medications 1 h before or 4 h after cholestyramine intake
- High doses of cholestyramine (greater than 32 g/day) may be associated with malabsorption of fat-soluble vitamins and hemorrhagic diathesis and osteomalacia

# Take Home Message

- Careful history can narrow down your diagnosis in dysphagia and chronic diarrhea
- Early endoscopy referral in certain cases will significantly improve patient long term management
- Empirical treatment with Bile Acid Sequestrant might be helpful after colonoscopy has ruled out other causes
- Brief explanation of how to take medications could go a long way with patient compliance and symptoms

# References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- *Nirmala P. Gonsalves, MD, Seema S. Aceves, MD, PhD Journal of Allergy and Clinical Immunology* Volume 145 Issue 1 Pages 1-7 (January 2020)

# Thank You!