Gastrointestinal Dimensions: Eating Disorders/Malnutrition

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Disclosures

None except:

• Bias toward our critical responsibility and value of effective sustained nutrition support

• Laced together with compassionate early psycho-behavioral intervention
Session Objective

• By the end of this session, participants will be able to:
  • Understand Malnutrition effects on GI Organ System tissue and function
  • Understand Specific Eating Disorder GI comorbidities
  • Understand GI Disorders mimicking or complicating Eating Disorders
  • Review an Approach to GI dysfunction in Eating Disorders: diagnosis, prevention and treatment
  • Know when to consult the GI specialist
Eating Disorders DSM-5

- Anorexia Nervosa (AN)
- Bulimia Nervosa (BN)
- Binge Eating Disorder (BED)
- Pica
- Rumination
- Avoidant/Restrictive Food Intake Disorder (ARFID)

Also:
- Many Mixed forms
- EDO-NOS
- Secondary DOE
“Eating Disorders are 90% Mental...
The other half is Physical”
Marasmus

Gk: withering
Effects of Starvation

- Food preoccupations/Food hoarding
- Abnormal taste preferences
- Binge eating
- Depression
- Obsessional behavior
- Apathy/irritability
- Inability to respond to feedback signals
Starvation Effects

- Study by Ancel Keys, 1944-5, Minnesota
  - Conscientious objectors to Korean Conflict
  - 32 healthy men of draft age “volunteers”
  - 3 months normal nutrition baseline
  - 6 months @ 50% intake; 25% weight loss
  - Altered Eating Behaviors:
    - Food preoccupation: ritualistic
    - Hoarding behavior
    - Gum chewing
    - Binge eating after experiment ended
    - Anxiety/Obsessive behaviors
Starvation and the Brain

• Demonstrated atrophy of the starved brain

• Alterations in brain function:
  • Adaptive responses: conserve energy, increase nutrient intake.
  • Maladaptive responses:
    • Alterations in neurochemistry
    • Enhanced behaviors/traits

• Acute vs. Chronic undernutrition
Sustained Hypocaloric Intake

- 3 week hypocaloric (1000 kcal) diet
- 6 men and 6 women lost 6% and 4.5 % body weight
- L-Tryptophan infusion at 0 and 3 weeks
- TRH injection after each LTP infusion
- Prolactin measured after infusions,
  - Bio-marker for 5HT receptor status
  - Dramatic rise in women, not men at 3 weeks
- Consider upregulated 5HT receptors in dietary restriction in women
  - 5 or more 5HT receptors are satiety receptors.
  - Dieting reduces synaptic 5-HT:
    - Short term effect of increased appetite
    - Long term effect of upregulated receptors \(\rightarrow\) increased satiety response
    - Inability to increase intake despite appetite

Before After Before After

Female Male

Prolactin AUC

Before After Before After

Male

Female
Food-Restriction Induced Hyperactivity

• Rats restricted to chow for 90 mins./d lose weight and stabilize.
• Access to running wheel → compulsive running → lethal weight loss
• A model: OCD, hyperactivity and/or AN ?
  • Motor activity → increases 5-HIAA, catecholamines
• Fluoxetine attenuated the FRIH:
  • serotonin receptor mediated
• No response to imipramine
  • Altemus M et al. Pharm Biochem Behav 1996;53:123-31
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Functional Gastro-Intestinal Disorders (FGID)
(Symptoms arising from the GI tract not explained by any detectable disease)

**Esophagus**
- Functional heartburn
- Gastro-Esophageal Reflux
- Functional Dysphagia
- Globus

**Gastro-Duodenal**
- Functional dyspepsia
- Belching Disorders
- Nausea and Vomiting Disorders

**Gallbladder/Sphincter of Oddi**
- Functional gallbladder disorder
- Functional SO disorder

**Functional Bowel Disorders**
- Irritable Bowel Syndrome
- Functional Constipation
- Functional Bloating
- Functional Diarrhea
- Functional Abdominal Pain

Drossman DA. Gastroenterology 2006;130:1377-90
GI disease/dysfunction Meets EDO Behavior

1. GI disease/dysfunction $\rightarrow$ EDOB $\rightarrow$ Malnutrition
   - Celiac, Crohn Disease, Irritable Bowel Syndrome
2. GI disease/dysfunction $\rightarrow$ Malnutrition $\rightarrow$ EDOB
   - Cystic Fibrosis, Crohn Disease, Type I DM/gastroparesis
3. EDOB $\rightarrow$ GI disease/dysfunction
   - Constipation-IBS, Oral-Esophageal injury, Gastric Distention/gastroparesis
4. EDOB $\rightarrow$ Malnutrition $\rightarrow$ GI disease/dysfunction
   - Fatty liver disease; villus atrophy, Sup. Mes. Art. (Duodenal Obstruction) Syn.
GI DDx of “Eating Disorder Patient”

• Inflammatory disease: Celiac disease, IBD (Crohn’s or UC)
• Chronic hepatitis, Chronic pancreatitis, Cystic Fibrosis
• Esophagitis- Eosinophilic
• Gastroparesis- idiopathic, post-viral, CGD, Myopathy, Type 1 DM
• Irritable Bowel Syndrome- stool retention
• Other: Addison’s, Lymphoma/Leukemia

*Screen patients for primary GI conditions that mimic EDO’s*
Presentations: GI conditions in EDO

- Dysphagia/Pyrosis: **acid reflux** vs eosinophilic esophagitis
- Epigastric pain: Esophagitis, gastritis, **dyspepsia**, nocturnal: PUD, pancreatitis, cholecystitis
- Supraumbilical pain: **Stool retention**, IBD- ileitis/proximal colitis, lymphoma
- Infraumbilical pain: **Constipation**, diverticulosis/itis, IBD- distal colitis
- Back pain: Scapula/shoulder: pancreas/biliary; post- duodenal ulcer, UTI/pyelo/stone; lumbosacral constipation
- Early satiety: Chronic stool retention(cologastric inhibition); gastroparesis (post–viral vs metabolic); small intestinal distention due to gas/distention consequences: “bloating, cramping, urgency”; IBS- irritable bowel syndrome- (diarrhea vs constipation dominant)
- Nausea/Vomiting: gastroparesis, GERD, Hiatal Hernia, Superior Mesenteric Artery syndrome, Stool Retention:ascending/transverse colon; Acute gastric distention
- Diarrhea, gas, distention, pain: **lactose intolerance**(lactase deficiency vs. post-viral); celiac disease, giardiasis/cryptosporidium; HIV, post-viral enteropathy
- Inflammatory: fever, chills, rash, oral/perianal lesions, blood, nocturnal,mucus-urgency/frequency of stools vs. tenesmus and “Crohnstipation”
GI tract changes in general undernutrition:

- Motility:
  - Cephalic phase: *dysregulated appetite*
  - Oropharyngeal initiation: *rare*
  - Esophageal peristalsis/sphincter function: *preserved*
  - Gastroparesis – slowed gastric emptying - atrophy-distention
    - Gastric Emptying: *solids delayed*
  - Gastrocolic response: *with gastroparesis* - *is impaired*
  - Cologastric inhibition: *stool retention/colonic distention*
  - Prolonged intestinal transit
  - Stool Retention/constipation

- Lumenal Digestion:
  - Hypochlorhydria
  - Pancreatic insufficiency
  - Biliary insufficiency

- Mucosal Digestion:
  - Brush border membrane
  - Small bowel bacterial overgrowth

- Mucosal Absorption:
  - Crypt hypoplasia, Villus atrophy
  - Epithelial disruption/dis-integration-increased permeability

**Consequence:** Reduced Nutrient intake and Assimilation

*Most reverse or improve with nutrition*
Nutrition/EDO Related Acquired GI Pathology

• Oral:
  • Dental erosions, caries, -enamel loss/polished surface.
  • Gingival periodontitis: rare in children- palatal ulcers –Vitamin C
  • Stomatitis: cheilosis- angular stomatitis: iron, zinc, B2
  • Nutritional deficits:
    • Vit C: gingivitis/bleeding (scurvy)
    • B vitamins(B1-3,6,12): mucosal atrophy, atrophic glossitis, glossodynia(burning tongue)
    • Oral candidiasis(thrush)
  • Sialadenosis: salivary gland(acinar) hyperplasia: parotitis→necrosis
• Rx: PPI treatment, consider motility agent for gastric emptying, manage stool retention that slows gastric emptying– observe for 2 hours after scheduled meals.
• Esophagus:
  • Pyrosis (heartburn), chest pain, dysphagia complaints prevalent
  • GERD: Acid/Pepsin exposure: esophagitis—metaplasia→dysplasia (Barrett’s)
  • Esophageal motility usually normal despite c/o dysphagia
  • Mallory Weiss tears—rare rupture (Boerhaave syndrome) with BN

• Stomach:
  • Gastroparesis vs Functional dyspepsia
    • Impaired fundus accommodation vs antral hypomotility
    • Gastric bezoar
    • Muscle atrophy
    • Autonomic dysfunction
  • Acute gastric distention—rupture rare
• Small Intestine:
  • Prolonged gastro-cecalt transit
  • Superior mesenteric artery syndrome: Rx: Transpyloric (NJ) feeding
  • Absorption – relatively preserved

• Large Intestine:
  • IBS-Constipation– may persist after restored nutrition
  • Anorectal/pelvic floor dysfunction prevalent 42% AN
  • Rectal prolapse[DDx: celiac, cystic fibrosis]
  • Laxative abuse– less risky unless castor oil or anthraquinones (senna and cascara) melanosis coli staining
  • Necrotizing Colitis
  • Rx: PEG 3350(Miralax) bowel prep cleanout→ sufficient prn to maintain daily complete BMs→ link toilet to meal—short walk stimulation.
• Pancreas:
  • Fibrosis in general with Protein-Energy Malnutrition—atrophy
  • Reduced amylase, lipase, trypsin (but normal bicarbonate excretion)
  • Reports of acute pancreatitis – not chronic—
    • Confounders: elevated serum amylase levels may be salivary origin
    Elevated serum lipase is seen in diabetic ketoacidosis—or ketosis.
  Rx: test fecal elastase, stool qualitative fat—consider pancrelipase

• Liver:
  • Transaminase elevation—improves with refeeding
  • NAFLD – hepatic steatosis→steatohepatitis
  • Starvation induced autophagy of hepatocytes and liver failure
  • Rx: Consider Vitamin E 800 IU supplementation for high ALT
Approach:

- Eating Disorder Phenotypes overlap with Chronic GI phenotypes:
  - Much of EDO Behavioral and GI dysfunction will improve with nutrition
- EDO does not logically exclude underlying or associated GI disorder
  - Cause, Consequence or Comorbidity
- Severe chronic malnutrition due to chronic energy deficit and/or somatic disease: can mimic EDO, including secondary psychiatric features of anxiety-OCD/depression and paradoxical anorexia related to disturbance of hypothalamic satiety regulation.
- Nutrition Support is a critical objectively measurable effective intervention related to prognosis.
- Cognitive based and pharmacologic based interventions may depend on restored brain-body nutritional and metabolic status.
Pharmacologic Interventions: Little Data

• Cyproheptadine-anti-histamine/anti-serotonin: Appetite stimulation
  • Also increases gastric accommodation/relaxation/reduce vomiting
• Zinc supplementation: increases NPY and orexin: 1 RCT-increased BMI
• Vitamin B12, Selenium: if plasma levels low
• PUFA-omega 3 and 6: may help
• Motility: metoclopramide, erythromycin, azithromycin, domperidone
• Acid suppression: PPI
• Laxatives: non-irritant osmotic laxative - polyethylene glycol, lactulose
• Probiotics: Hypothesis
When to Consult GI

• When Symptoms do not respond to Nutrition Support
• When Nutrition support cannot be provided due to GI symptoms
• For red flags of hematemesis, hematochezia, abdominal distention, persistent pain or tenderness, nocturnal pain, odynophagia or dysphagia.
• For positive celiac screen, occult blood, lactoferrin/calprotectin, elevated ESR/CRP in setting of GI dysfunction
References:

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NEXT SESSION:

June 20, 2019
12:00-1:30 p.m.

Outpatient Nutrition Care for Patients with Eating Disorders

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