12/23/25 Morning Report with @CPSolvers

"One life, so many dreams":--> "M Leaf Diagnosis" **Case Presenter**: Kirtan (@KirtanPatolia) **Case Discussants**: Dr. Gurpreet Dhaliwal

https://clinicalproblemsolving.com/present-a-case/

Scribing (Eyron)

CC: 80/F p/w 2 months of postprandial abdominal pain, nausea, vomiting and 20 lb weight loss.

HPI: Abdominal pain is progressive, cramping moderate, diffuse, lasting for 30 minutes at a time then resolves, scared of eating 2/2 symptoms hence developed weight loss. Also reports some anorexia. Abdominal pain, n/v was not worsening, occurs once daily.

ROS: No fevers, chills, night sweats, hematochezia, constipation, diarrhea, arthralgias, rashes, cough, chest pain, No dysphagia, odynophagia.

PMH:
HTN
Aortic stenosis
Meds:
Amlodipine

Fam Hx: NC

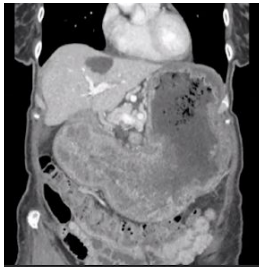
Social Hx: No EtOH, smoking

Health-Related Behaviors: From St. Louis

Allergies: None

Vitals: T: nl **HR**: nl **BP**:nl **RR**: nl **Sat**: nl **BMI**:
Exam: Abd: Generalized mild abdominal tenderness, no rebound or rigidity
Otherwise nl

Notable Labs & Imaging:
Hematology:
CBC wnl
Chemistry:
CMP wnl Alb 2.5-3 LFTs wnl
INR aPTT wnl | TSH, HIV, Syphilis wnl |
UA wnl | B12, folate, Vit K, Vit D, ferritin wnl
Anti-TTG and anti-Gliadin (+)
Fasting serum gastrin wnl
Imaging:



CT A/P: Gastric rugal thickening, no evidence of LAD | CT Chest: wnl
EGD + Duodenal Bx: Foveolar hyperplasia(gastric glands that secrete acid is hyperplastic)
in prominent gastric folds. No evidence of H. pylori or neoplastic cells. Duodenitis with neutrophilic infiltration and villous atrophy -> c/w hypertrophic gastropathy and Celiac disease

Tx with gluten free diet for 4 months w/o improvement of symptoms and persistent weight loss. Still with postprandial abdominal pain and weight loss (35 lbs since onset). Now associated with **painless hair loss and painless nail loss**. Repeat labs showed **low ALP**

Further Workup:
ANA, anti-goblet cell wnl | Zinc + Copper low | Repeat anti-TTG and anti-Gliadin (+)
CT A/P: Extensive mass like thickening of stomach with polypoid lesions situated near the antrum causing GOO, no LAD, liver/spleen wnl, colon, jejunum, ileum wnl
EGD/Colonoscopy: Multiple gastric polyps almost obstructing the entire lumen and gastric outlet, erythematous mucosa, duodenal villous atrophy | Colonoscopy wnl
Bx: Inflammatory/hyperplastic polyps with lymphocytes, eosinophils, no malignant/dysplastic cells | Staining negative

Dx: Cronkhite-Canada Syndrome

Problem Representation: 80/F p/w abdominal pain, n/v, and weight loss x months. Found to be anti-TTG and anti-Gliadin (+). CT revealed gastric rugal thickening and EGD with biopsy showed duodenitis with neutrophilic and villous atrophy c/w Celiac disease. Patient treated with gluten free diet x months without improvement of symptoms now a/w hair and nail loss. Found to have low Zn/Cu. Repeat EGD with biopsy showed inflammatory polyps w/ lymphocytes/eosinophils c/w Cronkhite-Canada syndrome

Teaching Points (Manaswini)

Approach to Abdominal Pain: Hold off adjective of the abd pain until we get general framework right! (For example: if fever - is it neutropenic fever or due to travelling/ HIV fever). We know the recurring VIPO mnemonic

- Postprandial pain- Think Proximal GI structures involvement (GERD, dyspepsia, ulcer, chronic pancreatitis, biliary system VS not distal (like colon xx)
- Degree with which the pain lasts- time course is crucial in all cases, and keep a lookout for how the case evolves
- Fun fact: Aortic stenosis too has predilection for atherosclerosis!

What do we think of if Patient has access to food but does not eat it? - 1. Anorexia(lack of desire) 2. Dysgeusia(lack of taste) 3. Dysphagia(if they can taste and have the will) 4. Abdomen related(mesenteric ischemia, SMA syndrome)
Patients with mesenteric ischemia may eventually stop reporting pain because they adapt by reducing food intake, having learned that eating less alleviates symptoms; over time, this behavioral adjustment may lead them to perceive themselves as pain-free→analogous to reduced symptom reporting of dyspnea in patients with coronary artery disease.

- Weight loss can be due to an anabolic issue (eating less) vs catabolic(cancer)

What is infiltrating the GI tract? –1. Slow moving cancer(due to age,weight loss)-Linitis plastica(gastric adenocarcinoma-type 4)/ lymphoma[@ileum & stomach]/maltomas(H.pylori) 2. Cellular based infiltration like-eosinophilic gastritis, sarcoidosis, amyloidosis, mastocytosis, histiocytosis, (NE tumours less likely) → Do a flow cytometry 3.Infection:TB- Leading worldwide(gastric aspirates were done before in pediatrics), H.pylori, PAS for Whipple's disease 4. Inorganic substances depositing in tissues
Approach to low Albumin- points ascribed to it: 1. Nutrition 2. Edema(urinary loss, liver disease, protein losing enteropathy)

- Young children: Congenital lymphatic disorder , Adults: What is infiltrating/interrupting the lymphatics – to cause protein losing lymphadenopathy;; Albumin: -ve phase reactant which is a marker of inflammation(malnutrition)

Serologies that can be pursued: Celiac disease, (Anti-goblet cell/enterocyte Ab's) in autoimmune enteritis
What drives the thickening of stomach wall ? -1. ZES(excess gastrin),2.Malignancies at the gastric outlet (e.g.antrum) may be missed on superficial sampling; therefore,**deep biopsies are essential for accurate diagnosis of cancer there**, duodenitis(villous atrophy-seen in celiac sprue syndromes→*lymphocytic* infiltration is more common in celiac).

- We have to find out if it is a Lymphoma activating the immune system here.

Non scarring alopecia- 1. Syphilis(systemic disorders)
2. Metabolic or nutritional deficiency (**Zn: is a cofactor for ALP**→ hence Zn def causes low ALP}- also seen in Wilsons! [Important to think why is there Zn deficiency? →a vicious cycle leading to Acrodermatitis enteropathica]. - Fact: Excess Zn → decreased Copper Age might be making us lean away from celiac disease. Twice +ve celiac antibodies indicates celiac but it may not reflect underlying disease, or Celiac transforming into a T- cell lymphoma→ may be in jejunum or ileum, where endoscopy results might be -ve because the scope cannot reach there)
In this Case: 1. The disease here is localised to the stomach→Lack of Ileal disease and Local Lymph nodes make us not think of lymphoma. 2.Not T Cell lymphoma from Celiac as it affects the intestine, not the stomach like here 3.Not ZES→ as more thickening and ulceration seen, not polyps. - So, think in the lines of Polyposis syndromes or if pt has Excess GH(acromegaly→proliferates tissue causing polyps). Non malignant in nature- here, so also think autoimmunity(ANA+ve conditions/vasculitis/myositis/S-veSA)> infection

- Think **Cronkhite Canada syndrome**: Presents with prominent Gastric polyps, inflammatory polyps and **inflammatory INTERVAL(mucosa btw 2 polyps mucosa on histopath)**, onychodystrophy, alopecia, protein losing enteropathy.

→Acquired syndrome(hence presents late)--> **NON GENETIC**→ it is Immune mediated, so Extremely important that we give steroids, cyclosporine/AZA→if not working, give TNF alpha inhibitors [in addition to the mainstream surgery- removing stomach, colon, polypectomy]
This patient had Celiac as well but did not respond to gluten free diet because of underlying Cronkhite Canada syndrome.
Why is there hair and nail changes? → Is it Zn def due to celiac or is it from Cronkhite Canada syndrome?: Ectodermal structure damage(underlying inflammation/nutrient deficiency)→ hair and nail changes
Important to note that a person with celiac and low Zn levels mostly will not have hair and nail changes. Inflammatory nature of Cronkhite Canada syndrome causes development of these changes(i.e the underlying multifaceted vulnerability)
(Note: Nails and hair changes → mimic Menetriers, hypertrophic gastropathy)

Art of Vocabulary, Logic, Rhetoric: make the foundation of a Professional →Talk, think, speak & write like a doctor. Do check out the excellent Teaching Points slides by Dr. Kirtan Patolia at the end of the VMR!