

Micronutrient Deficiency in IBD; An Underappreciated Complication



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Case Description

History of Present Illness:

PR is 61-year-old female with history of endometrial cancer status post hysterectomy, ulcerative colitis recently started on Entyvio, and traumatic splenectomy who presented to Maine Medical Center with six month of weakness, failure to thrive, diarrhea, and weight loss.

She reported ongoing poor oral intake and had lost 60lbs over that six-month period. She described a several-week history of watery, nonbloody/nonmelanous diarrhea with intermittent nausea and cramping, but no profound abdominal pain. She also reported some exertional fatigue, tiring after climbing three flights of stairs. She also admitted to emotional challenges after the passing of her mother five months prior to admission.

Social History

Lives in Rangeley, ME with husband. No children No tobacco or EtOH use Occasional marijuana, 3-4x/week

Medications

Vedolizumab

Prednisone

Dicyclomine

Ondansetron

Mirtazapine

Potassium Chloride

Physical Exam:

BP 98/66 | HR 104 | T 36.9 °C | Resp 16 | Wt 58.7 kg | SpO2 100%

General: Awake, alert, oriented. No distress. Lying in stretcher Eyes: No scleral icterus. No conjunctival injection

HENT: Dry membranes moist. No oral lesions. Normocephalic, atraumatic.

Neck: No lymphadenopathy or masses. No nuchal rigidity.

Cardiovascular: Regular rhythm, tachycardic. No murmurs. 2+ pulses Pulm: No accessory muscle use. Clear to auscultation bilaterally with good air entry.

GI: Non-distended. Normoactive bowel sounds. Soft, non-tender. No guarding, rebound or rigidity

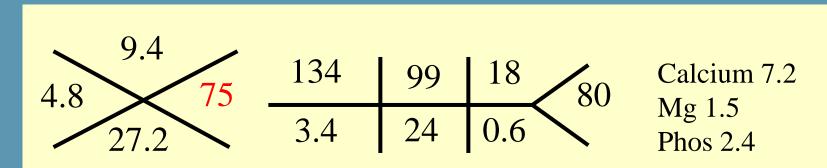
Musculoskeletal: Full painless range of motion. No joint swelling or erythema.

Neurological: Cranial nerves II-XII intact. No focal deficits.

Skin: No rashes or lesions. Trace peripheral edema

Psych: attention normal, thoughts organized, short term memory intact

Laboratory Data



Protein 5.0 Albumin 1.8 Bilirubin 1.2

MCV 97.1

AST 8 ALT 21 Alk Phos 92 INR 1.4

CRP: 100.1

ESR: 1

Calprotectin 296 C diff: negative Stool cx: negative Stool O&P: negative TTG Ab:1.2

Vit B12: 1239 Iron 55 Vit A 5.2 (32-78) Vit E 7.7 (5.5-17) Vit C < 0.1 (0.4-2.0) Copper 334 (810-1990) FOBT: positive Zinc 33 (60-130)

Vit D: 33

Colonoscopy

3 Appendiceal orifice/cecum

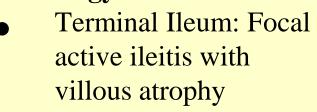


4 Terminal ileum

Endoscopic Findings Mild erythema of

- terminal ileum Pseudopolyposis throughout ascending, transverse, and
- descending colon Intervening mucosa without erythema, ulceration
- No evidence of active colitis
- Sparing of rectum and rectosigmoid colon

Pathology



- Ascending/Transverse Colon: mild/moderate chronic active colitis with cryptitis and crypt abscess formation Descending colon:
- Chronic inactive colitis Rectum/Sigmoid: Chronic inactive colitis

Micronutrient Deficiencies

Micronutrient	Pathophysiology	Symptoms	Prevalence
		Peripheral Neuropathy	CD: 32%
B1 (thiamine)	Unclear	Cardiomyopathy	UC: Unknown
		, , ,	
	Poor Intake	Megaloblastic Anemia	
	•	Atrophic Glossitis	
DO (folato)	Medications (MTX,		CD: 0 3C0/
B9 (folate)	Sulfasalazine)	Depression	CD: 0-26%
		Megaloblastic Anemia	CD: 11-22%
		Pancytopenia	> 60cm ileal resection:
	Ileitis	Peripheral Neuropathy	100%
B12	Ileal Resection	Dementia	20-40cm resection: 48%
		Poor Wound Healing	
		Gingivitis	
		Scaly Skin	
С	Poor Intake	, Arthralgias	CD: > 50%
	Door Intaka	Door Mound Hooling	
		Poor Wound Healing	
	Fat Malabsorption		IDD: 0 440/
A	Bile Salt Deficiency	хегоринантна	IBD: 0-44%
		Neuropathy	
	Fat Malabsorption	, ,	
E	Bile Salt Deficiency	Anemia	Unknown
	Poor Intake		
	Fat Malabsorption		CD: 22-70%
D	Decreased Sun	Osteopenia	UC: 45%
		Abnormal Bone	
		Metabolism	
	Poor Intake	Bleeding	
	Fat Malabsorption		Unknown
K	Bile Salt Deficiency		CD > UC
		100000000000000000000000000000000000000	
	Vitamin D	Docroscod Rono Donsity	
	Vitamin D	Decreased Bone Density Hypoparathyroidism	
	•	Hypoparathyroidism	
	Hypomagnesemia Poor Intake	Hypertension Muscle Cramps	
Calcium	Steroid Use	Prolonged QTc	IBD: 80-86%
Calcium	Poor Intake	Bone Health	טט. טט טטאט. טעוו
Magnosium			Lincloor
Magnesium	Diarrheal Losses	Hypocalcemia	Unclear
	Chronic Blood Loss	Microcytic Anemia	
	Poor Intake	Fatigue	
	Impaired Iron	Glossitis	
Iron	Metabolism	Restless Leg Syndrome	IBD: 36-90%
		Poor Wound Healing	
	Chronic Diarrhea	Acrodermatitis	
Zinc	Malabsorption	Poor Taste	Unclear
	,		
		Cardiomyopathy	
		Cartilage Degeneration	Unknown
Selenium	Post-TPN	Hypothyroidism	Low in both UC/CD
SCICITION			LOW III DOUT OC/CD
Copper	Malabsorption Zinc Ingestion	Neuropathy Pancytopenia	Unknown
CODDCI	LIIIC IIICCSUUII	II GIICY LODCIIIG	

Hospital Course

- Patient was well-appearing on admission, but quickly deteriorated over her two-week course
- Infectious etiologies were ruled out
- Colonoscopies showed mild to moderate colitis, patient remained on IV steroids
- Due to initial concerns for food aversion and depression, enteral feeds were held for fear of insurance coverage
- Labs ultimately revealed severe macro- and micronutrient deficiencies, enteral feeds were initiated but poorly tolerated
- Patient suffered nutritional hypoglycemia requiring dextrose fluids
- Patient had ongoing pancytopenia leading to bone marrow biopsy which showed no evidence of malignancy
- Prior to TPN initiation, the patient suffered a small bowel perforation \rightarrow shock \rightarrow transition to comfort care

Clinical Implications

Given ileal findings on colonoscopy, profound malnutrition, and small bowel inflammation/fistula on CT, Crohn's disease was the most likely etiology despite her previous diagnosis of ulcerative colitis. There are no definitive studies linking micronutrient deficiencies to bowel perforation, but anemia due to iron deficiency has been shown to worsen malabsorption, vitamin D supplementation has been shown to decrease hospitalizations, and vitamin B12 deficiency may increase the likelihood of surgery in Crohn's patients [3,6]. While unrecognized small bowel disease and glucocorticoid use likely lead to this patient's bowel perforation, it remains possible that nutritional deficiencies also contributed towards bowel thinning and eventual perforation. Although enteral nutrition was previously thought inferior to steroids in the treatment of adult Crohn's disease, recent evidence shows similar remission rates among the two therapies. Therefore, in addition to current biologic agents, early enteral nutrition should be considered as a means of targeting both inflammation and malnutrition in Crohn's disease. [4]

References

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