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Trends of Micronutrient Deficiencies in Inflammatory Bowel Disease

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Abstract

Background: Vitamin deficiencies associated with malnutrition in patients with inflammatory bowel disease (IBD) can lead to complications including anemia, coagulopathy, poor wound healing, and colorectal cancer. This study aimed to investigate micronutrient deficiencies (vitamins B1, B6, B12, and zinc) in IBD patients and highlight symptoms or historical features to aid in the recognition of micronutrient deficiencies.

Materials & Methods: A retrospective electronic chart review was performed on adults diagnosed with Crohn's disease (CD) or ulcerative colitis (UC) hospitalized for IBD flare at a tertiary care center between 1/2013 to 6/2017. Patients with serum or whole blood micronutrient levels were included. Pregnant and incarcerated patients were excluded. 356 IBD patients (285 CD, 71 UC) met the inclusion criteria. Micronutrients were assessed in a subset of IBD patients (B1: 30.6%, B6: 34.8%, B12: 97.2%, Zinc: 18.8%).

Results: Overall, 29.8% of patients had micronutrient deficiencies. The proportion of patients with B1, B6, B12, and zinc deficiencies were 22.6%, 41.3%, 10.4%, and 30.8% for CD and 25%, 35%, 3.0% and 40% for UC, respectively. The most common symptoms or historical features associated with micronutrient deficiency were peripheral neuropathy (B1, B6, B12), elevated inflammatory markers, steroid use or history of DVT/PE before admission (B6), edema (B1), anemia, cognitive impairment, myalgia, gait disturbance (B12), depression (B6, B12), diarrhea, decreased albumin, and bowel resection (zinc).

Conclusion: Micronutrient deficiencies are common in IBD patients, yet they are not routinely assessed. B1, B6, and zinc deficiencies are particularly underrecognized. Associated historical features should raise suspicion and prompt assessment and treatment.

Keywords: micronutrient, malnutrition, inflammatory bowel disease, vitamin deficiency

Introduction

Several early retrospective studies have shown that inflammatory bowel disease (IBD) is associated with malnutrition, particularly in patients with active, severe disease.[1-3] The pathophysiology behind this malnutrition is multifactorial: decreased food intake, increased intestinal loss, malabsorption, hypermetabolic state, drug interactions, and long-term parenteral nutrition can all contribute to nutrient deficiencies in this population.[1] While deficiencies may be related to both macronutrients (protein and energy intake) and micronutrients (vitamins and minerals), micronutrient deficiencies are more common in the adult IBD population and can occur even with relatively mild disease or during remission.[1] Vitamin and mineral deficiencies can ultimately lead to several other complications, including anemia, poor wound healing, osteoporosis, thrombophilia, and colorectal cancer.[1] In addition, malnourished IBD patients may be more likely to have prolonged hospitalization and higher mortality than IBD patients without nutrient deficiencies.[2]

The incidence of these deficiencies in hospitalized IBD patients is not clear based on the currently available literature. Therefore, a comprehensive study is needed to further define the incidence in this population. This study aimed to investigate micronutrient deficiencies (vitamins B1, B6, B12, and zinc) in IBD patients and highlight symptoms or historical features to aid in the recognition of micronutrient deficiencies.

Methods:

A single-center retrospective electronic chart review was performed on adults with an ICD-9/10 code diagnosis of Crohn's disease (CD) or ulcerative colitis (UC) during their index hospitalization for IBD flare to a tertiary care center between January 1st, 2013, and June 30th, 2017. Adults (>18 years old) with IBD with serum or whole blood micronutrient levels were included and standard serum or whole blood thresholds for each micronutrient were used. Pregnant and incarcerated patients were excluded.

Data Collection

Patients' age, gender, height, total body weight (TBW), ideal body weight (IBW), body mass index (BMI), IBD type, site of intestinal tract affected by IBD, history of fistula, strictures, or surgery. Whole blood thiamin and serum pyridoxine levels were measured with high-performance liquid chromatography-tandem mass spectrometry, whereas serum zinc level was measured with inductively coupled plasma-mass spectrometry (ARUP Laboratories, Salt Lake City, UT). Serum vitamin B12 levels were measured with DxI immunoassay (lab location was blinded for review purposes).

Statistical Analysis

Statistical software JMP (SAS v 16) was used to analyze the data. Descriptive analyses were applied for demographic variables and medical condition variables. Means, standard deviations, and ranges were calculated for continuous variables; frequencies and percentages were calculated for categorical variables. Results are reported in means and percentages.

Results:

During the study period, 1,115 unique entries were screened, and 356 patients were found to have an IBD (285 CD, 71 UC). This subset of 356 patients with at least 1 micronutrient level obtained and 106 (29.8%) had at least one micronutrient deficiency with a mean age of 45 years.

Table 1: Patient demographics

Characteristic	All Patients n = 356	Micronutrient Deficient patients n = 106	CD			UC		
			All CD patients n = 285	Non-deficient n = 196	Deficient n = 89	l UC patie n = 71	Non-deficient n = 54	Deficient n = 17
Age, y, mean (range)	48 (18 - 92)	45 (19 -79)	47 (19 - 92)	48 (19 - 92)	4 (19 - 79	52 (18 - 84)	52 (18 - 84)	53 (24 - 79)
Female, n (%)	187 (52.5)	64 (60.4)	156 (54.7)	92 (47.0)	60 (67.4)	34 (47.9)	31 (57.4)	4 (23.5)
Weight, kg, mean ± SD	73.5 ± 21.9	71.3 ± 23.2	73.9 ± 22.6	75.9 ± 21.9	0.4 ± 23.(	72.1 ± 19.0	70.5 ± 18.2	76.1 ± 21.1
Body mass index, kg/m^2; mean ± SD	26.0 ± 7.39	24.9 ± 7.61	26.15 ± 7.68	26.91 ± 7.53	4.86 ± 7.8	25.36 ± 6.1(	25.36 ± 5.94	25.31 ± 6.67
Length of stay, mean days (range)	10.0 (0 - 96)	8.6 (0 - 56)	10.6 (0 - 85)	11.0 (1 - 85)	.5 (0 - 56	8.2 (2 - 96)	9.3 (2 - 96)	5.3 (2 - 15)
Alcohol use, n (%)								
Yes	71 (19.9)	22 (20.8)	66 (23.2)	45 (23.0)	21 (23.6)	5 (7.0)	4 (7.4)	1 (5.9)
No	212 (59.6)	58 (54.7)	162 (67.8)	117 (59.7)	45 (50.6)	50 (70.4)	37 (68.5)	13 (76.5)
Not asked	73 (20.5)	26 (24.5)	57 (20.0)	34 (17.3)	23 (25.8)	16 (22.5)	13 (24.1)	3 (17.6)
Tobacco use, n (%)								
Yes/passive	50 (14.0)	14 (13.2)	44 (15.4)	32 (16.3)	12 (13.5)	6 (8.5)	4 (7.4)	2 (11.8)
Quit	93 (26.1)	24 (22.6)	70 (24.6)	51 (26.0)	19 (21.3)	23 (32.4)	18 (33.3)	5 (29.4)
Never	146 (41.0)	44 (41.5)	119 (41.8)	82 (41.8)	37 (41.6)	27 (38.0)	20 (37.0)	7 (41.2)
Not asked	67 (18.8)	24 (22.6)	52 (18.2)	31 (15.8)	21 (23.6)	15 (21.1)	12 (22.2)	3 (17.6)

Values mean (range) or n (%) unless otherwise noted.

CD, Crohn's disease; UC, ulcerative colitis; IBD, inflammatory bowel disease

(**Figure 1**). Sixty-four were females and 42 were males. Demographic characteristics of the study population are listed in **Table 1**. Twenty-one of 93 CD patients (22.6%) and 4 of 16 UC patients (25%) had thiamine deficiency. Forty-three of 104 CD patients (41.3%) and 7 of 20 UC patients (35%) had pyridoxine deficiency. Vitamin B12 was found to be deficient in 10.4% of CD and 3% of UC patients whereas Zinc was found to be deficient in 30.8% of CD patients and 40% of UC patients respectively

Table 2: Deficiency Incidence

Table 2-a: Summary of micronutrient deficiency Incidence among IBD patients

Incidence (%)		
	CD	UC
B1	22.6	25.0
B6	41.3	35.0
Zinc	30.8	40.0
B12	10.4	3.0

Table 2-b: Incidence of micronutrient deficiency among IBD patients

			Zinc	B1	B6	B12	Total	Total removing duplicates across micronutrients
Total entries								
		CD	78	113	152	587	930	
		UC	19	23	24	119	185	
		Subtotal	97	136	176	706	1115	
	Total unique entries							
		CD	52	93	104	280	529	285
		UC	15	16	20	66	117	71
		Subtotal	67	109	124	346	646	356
Unique deficient								
		CD	16	21	43	29	109	89
		UC	6	4	7	2	19	17
		Subtotal	22	25	50	31	128	106

(**Tables 2-a and Table 2-b**). **Figure 2a-d** illustrates yearly micronutrient deficiency during the study period. The most common symptoms or historical features associated with micronutrient deficiency were peripheral neuropathy (B1, B6, B12), cheilitis, elevated CRP, steroid use or history of DVT/PE prior to admission (B6), cardiomyopathy (B1), anemia, cognitive impairment, myalgia, gait disturbance (B12), depression (B6, B12), skin lesions, diarrhea, bowel resection, fistula or bypass surgery (zinc). Symptoms and clinical characteristics of each micronutrient deficiency, stratified by IBD type, are presented in

Table 3: Patient symptoms associated with deficiency

Zinc					**B6**		
	CD	**UC**				**CD**	**UC**
Total	**16**	**6**			**Total**	**43**	**7**
Zinc value, reference range (mcg/dL)	60 - 130				B6 value, reference range (mmol/L)	20 - 125	
Zinc value of deficient patients, mean ± SD (mcg/dL)	49.2 ± 7.8	47.4 ± 6.5			B6 value of deficient patients, mean ± SD (mmol/L)	10.6 ± 5.0	9.2 ± 5.9
Zinc value of non-deficient patients, mean ± SD (mcg/dL)	81.6 ± 17.2	72.1 ± 10.7			B6 value of non-deficient patients, mean ± SD (mmol/L)	46.2 ± 53.4	56.0 ± 41.7
Taste/Smell Alterations	3 (19)	0 (0)			Increased Inflammatory Markers (CRP, TNF-α, IL-6)	29 (67)	5 (71)
Skin Lesions	2 (13)	1 (17)			Alcoholism (>14 drinks per week)	0 (0)	0 (0)
Bowel Resection	6 (38)	3 (50)			Rheumatoid Arthritis	1 (2)	0 (0)
Diarrhea	12 (75)	4 (67)			Glossitis	1 (2)	1 (14)
Diarrhea >5x per day	5 (31)	1 (17)			Chelitis	2 (5)	2 (29)
Rash	7 (44)	0 (0)			Somnolence (Presented with)	2 (5)	1 (14)
Hair Loss	3 (19)	0 (0)			Depression (Presented with)	10 (23)	1 (14)
Fistulas	4 (25)	0 (0)			Peripheral Neuropathy	3 (7)	1 (14)

Albumin Deficient (<3.6 g/dL)	8 (50)	5 (83)			MI (history of)	0 (0)	1 (14)
In ICU During Admission	2 (13)	2 (33)			Ischemic Stroke (history of)	4 (9)	1 (14)
Infection	4 (25)	2 (33)			DVT/PE (history of)	9 (21)	3 (43)
Sepsis	1 (6)	1 (17)			Steroid use	22 (51)	0 (0)
B1					**B12**		
	CD	**UC**				**CD**	**UC**
Total	**21**	**4**			**Total**	**29**	**2**
B1 value, reference range (nmol/L)	70 - 180				B12 value, reference range (pg/mL)	243 - 894	
B1 value of deficient patients, mean ± SD (nmol/L)	26.0 ± 16.2	24.3 ± 13.1			B12 value of deficient patients, mean ± SD (pg/mL)	196.7 ± 51.1	217 ± 35.4
B1 value of non-deficient patients, mean ± SD (nmol/L)	135.7 ± 55.8	114.3 ± 40.6			B12 value of non-deficient patients, mean ± SD (pg/mL)	719.3 ± 464.7	835.5 ± 492.4
Peripheral neuropathy	5 (24)	3 (75)			Anemia	26 (90)	1 (50)
Cardiomyopathy (Beri Beri)	1 (5)	1 (25)			Megaloblastic anemia	1 (3)	0 (0)
Calf muscle tenderness	3 (14)	1 (25)			Pancytopenia	0 (0)	0 (0)
Edema	6 (29)	1 (25)			Peripheral neuropathy	16 (55)	1 (50)
Tachycardia	13 (62)	2 (50)			Paresthesias	13 (45)	2 (100)
Cardiomegaly	2 (10)	0 (0)			Ataxia	0 (0)	0 (0)
HF	3 (14)	1 (25)			Gait disturbances	3 (10)	0 (0)
CHF	2 (10)	1 (25)			Cerebral manifestations	0 (0)	0 (0)
					Cognitive Impairment	6 (21)	1 (50)
					Mental Changes	0 (0)	0 (0)
					Depression	12 (41)	0 (0)
					Psychosis	0 (0)	0 (0)
					Myalgias	9 (31)	0 (0)
					Paraplegia	0 (0)	0 (0)
					Dementia	0 (0)	0 (0)
Represented as n (%) unless otherwise noted							

CD, Crohn's disease; UC, ulcerative colitis; IBD, inflammatory bowel disease

Table 3. The most common symptoms or historical features associated with micronutrient deficiency were diarrhea and infection (zinc), depression (B6, B1), history of venous thromboembolism (B6), Tachycardia and edema (B1), anemia and peripheral neuropathy (B12)**.**

Discussion:

In this cohort of 356 IBD patients with 29.8% deficiency of at least one micronutrient, we collected associated symptoms related to each micronutrient to highlight the importance of detecting such deficiency and help guide clinicians in identifying such deficiencies. Incidence of micronutrient deficiency has been reported in more than half of IBD patients in one report and found to be 82.5% and 11.3% in CD and UC respectively in another. [2,4] Micronutrients play an important role in inflammatory signaling in addition to their antioxidant functions and cellular machinery processes. Thiamine is an important cofactor in cell ATP generation and aids in reducing cellular oxidative stress.[5] Vitamin B6 is a coenzyme in more than 140 transamination, decarboxylation, and transsulfuration reactions as well as in synthesis pathways for several neurotransmitters, such as aminobutyric acid, serotonin, dopamine, and glycine.[6] Additionally, it may help reduce inflammation and cellular oxidation. Vitamin B12 is involved in lipid, carbohydrates, and protein metabolism. Its deficiency may result in neurologic sequelae due to the inability to synthesize or maintain the lipoprotein myelin sheath. Zinc modulates immune function and helps with DNA, protein synthesis, and repairs. Additionally, it provides catalytic enzyme activities and supports intestinal barrier functions.

Underlying etiologies to such deficiencies include reduced oral intake, malabsorption, inflammation, increased energy expenditure, and increased losses.

Vitamin B1

Thiamine plays an important role in the catabolism of carbohydrates and proteins, mitochondrial ATP production, and reduction of cellular oxidative stress.[5] Thiamine is a water-soluble vitamin that is absorbed by passive diffusion and active transport in the Jejunum and ileum. Thiamine deficiency can lead to Wernicke encephalopathy (WE), Korsakoff psychosis, and neurological deficits such as beriberi, fatigue, irritability, poor

memory, sleep disturbances, or abdominal discomfort.[5] In our study the most common vitamin B1 deficiency symptoms we found were peripheral neuropathy (24% and 75%), tachycardia (62% and 50%), and edema (29% and 25%) for CD and UC patients respectively. In IBD patients, fatigue was shown to be the manifestation of thiamine deficiency.[7] The limited data currently available to support thiamine deficiency in IBD patients includes a case study of a UC patient with severe optic neuropathy and oculomotor palsy associated with symptoms showing improvements when treated with high doses of thiamine following deficiency diagnosis[8] and a case study of two non-alcoholic malnourished CD patients who developed Wernicke's encephalopathy following total parental nutrition without thiamine supplementation.[9] Supplementation following the WE diagnosis drastically alleviated symptoms.[9] A study examining CD patients in remission found that 32% had diminished levels of thiamine.[10] A trial with inactive IBD patients and severe chronic fatigue (displaying no other reason for fatigue) showed a significant benefit of high-dose oral thiamine.[11]

Vitamin B6

Pyridoxal 5'-phosphate (PLP) is the biologically active form of Vitamin B6 and is absorbed in the Jejunum and ileum. PLP is involved in a multitude of metabolic pathways, serving as a cofactor for over 140 biochemical reactions.[6] Severe vitamin B6 deficiency can be uncommon due to its absorption potential throughout the small intestine. However, PLP is seen to be reduced in individuals with inflammatory conditions and patients with high CRP.[12] Medications that may impact B6 absorption are diuretics and pyridoxine antagonists, such as theophylline, isoniazid, and penicillamine, as well as dialysis and states of overutilization such as pregnancy.[13] In our study the most common vitamin B6 deficiency symptoms we found were increased inflammatory markers (67% and 71%), depression (23% and 14%), deep vein thrombosis or pulmonary embolism history (21% and 43%) for CD and UC patients respectively.

Saibeni et al. found lower PLP concentration in CD and UC patients, especially those with active disease, compared to controls.[14] In a prospective analysis of micronutrient status in 93 patients with IBD, low vitamin B6 was found in 14% of patients, correlating more to CD patients with a stricturing phenotype ($p = 0.001$) than those with an inflammatory phenotype.[15] An assessment of 126 adults with IBD showed vitamin B6 deficiency in 29% of patients.[16] Although diet could be inferred as the potential cause of the higher prevalence of B6 deficiency in these patients, only 6% consumed inadequate amounts of vitamin B6, indicating dietary B6 is an unlikely cause of this deficiency.[16] This also applies to malabsorption as the incidence for CD and UC were around the same.[16] Inflammation may be the explanation for low B6 levels observed in IBD, supported by the fact that patients with rheumatoid arthritis, a disease also associated with inflammation, showed lower than normal PLP levels. Treatment includes 50-100 mg of pyridoxine per day.[6,13]

Vitamin B12

Vitamin B12 serves as a coenzyme in multiple biochemical reactions, including folate metabolism, DNA synthesis, RBC formation, and nerve function. Vitamin B12 is bound to intrinsic factors and absorbed in the distal ileum.[18] Common symptoms of a vitamin B12 deficiency include glossitis, neurological symptoms, and macrocytosis.[19] IBD is commonly associated with vitamin B12 (cobalamin) deficiency. Since absorption of vitamin B12 occurs in the small intestine, CD patients are at an increased risk of deficiency. In our study, the most common vitamin B12 deficiency symptoms we found were anemia (90% and 50%), peripheral neuropathy (55% and 50%), and paresthesia (45% and 100%) for CD and UC patients respectively. A longitudinal study of 180 CD patients showed the prevalence of B12 deficiency to be 15.6% compared to 2.8% in UC patients. [17] Ileal resection was shown to be a risk factor in developing a vitamin B12 deficiency. However, some argue that there is insufficient evidence to attribute a higher risk of B12 deficiency in CD patients based on ileal resection. [20] An assessment of 126 adults with IBD showed vitamin B12 deficiency in 18.4% of patients, with 4.8% consuming inadequate amounts of B12.[16]. The median serum B12 was significantly lower in CD than in UC patients. [16] In a study using data from multiple databases, the mean serum B12 concentration was found to be lower in Asian patients than in controls.[21] This data simply reinforces the monitoring of this vitamin in patients with IBD. Patients with IBD should be tested for B12 deficiency annually. [22] Oral cyanocobalamin proved to be an effective treatment for B12 deficiency in CD patients.[23]

Zinc

Zinc is a trace element that aids in the immune response, acting as a catalytic cofactor, and is vital for wound healing, and protein and collagen synthesis.[1] Zinc plays a pivotal role in cell turnover, repair systems, supporting the catalytic activity of > 300 enzymes, and the immune response. Zinc is absorbed in the small intestine, primarily in the duodenum and jejunum.[24] Zinc is a common deficiency among patients with IBD, during both active inflammation and remission phases.[16,25-26] In our study the most common zinc deficiency symptoms we found were diarrhea (75% and 67%), and albumin deficiency (50% and 83%) for CD and UC patients, respectively.

In a specific assessment, zinc was significantly lower in CD than in UC.[27] When solely observing CD patients, the prevalence of zinc deficiency was higher compared to controls, yet no significant difference in zinc serum concentrations was shown.[28] Decreased serum zinc concentration has the potential to enhance inflammation through a variety of pathophysiological mechanisms, some of which include increased proinflammatory cytokines and altered musical immunity. In CD, zinc plays the role of reducing the trans-mucosal leak by decreasing the number of inflammatory cells and reducing proinflammatory cytokine production.[28-29] In an analysis of patients enrolled in a prospectively collected IBD registry with at least two serum zinc measurements, those with a diagnosed zinc deficiency showed an increased risk of subsequent hospitalizations, surgeries, and disease-related complications.[30] These outcomes subsided with the normalization of zinc levels, serving as useful support for the close monitoring and compensation of zinc-deficient IBD patients. Zinc deficiency has been shown to be an indicator of shorter remission type between active flares, particularly in patients with CD.[15] In active macrophages, zinc suppresses nitrous oxide and prevents the production of reactive oxygen and nitrogen species. It is still a question, however, if this deficiency is a cause or an effect of IBD. USDA recommends zinc intake for men to be 11 mg per day and women to be 8 mg per day.[1] Standard supplementation for zinc is 220 mg of zinc sulfate or 25-50 mg of elemental zinc twice per day,

while patients with significant diarrhea are recommended to take 20-40 mg per day of zinc gluconate.[1] A potential cause of copper deficiency is prolonged zinc supplementation in zinc-deficient patients,[31] so supplementation should be stopped after 2-3 weeks.[1]

Limitations:

By design, this is a retrospective study with an inherent selection bias that limits its generalizability. Additionally, no cause-and-effect can be drawn from the study results. Moreover, sample size, especially for UC may limit the interpretation of the results. However, we collected associated symptoms with each micronutrient to help guide clinicians in identifying potential deficiencies. Although data was presented for indicators of other micronutrients deficiency, such as anemia for iron, and some patients studied were deficient in multiple micronutrients, this study reported on 4 micronutrients, and a separate study reports on an additional 5 micronutrients in this patient population. We cannot rule out confounding multiple deficiencies in the symptoms and characteristics presented as they were not isolated in this study. Correlation and causation of the symptoms assessed in this study cannot be established due to its retrospective nature and lack of temporal relationship as to when a patient was found to be deficient. Furthermore, micronutrient levels may also be affected in the setting of inflammation being acute phase reactants and may have impacted incidence data in our hospitalized study population.

Conclusions:

Active IBD has been associated with reduced oral intake, maldigestion, malabsorption, anorexia, increased loss due to vomiting, diarrhea, or increased output and weight loss. Micronutrient deficiencies are common in IBD patients, yet they are not routinely assessed. B1, B6, and zinc deficiencies are particularly underrecognized. Factors contributing to micronutrient deficiencies in IBD patients include reduced absorption, chronic inflammation, reduced intake, drug interactions, and prior surgeries. Associated historical features should raise suspicion and prompt assessment and treatment.

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