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Measurement of Nutrition Status in Crohn's Disease Patients Receiving Infliximab Therapy

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Abstract

Aim—There is limited information on the nutrition impact of antitumor necrosis factor- α treatment in adult Crohn's disease (CD). This study was performed to examine the effect of a 6-month course of infliximab on enterocyte function, nutrient status, metabolism, and body composition in these patients.

Methods—Seven CD patients were assessed for disease activity, enterocyte function, and body composition prior to, after 6 weeks, and after 6 months of infliximab treatment. Measurements included (1) disease activity: Inflammatory Bowel Disease Questionnaire, Harvey Bradshaw Index, and C-reactive protein; (2) enterocyte function: folate, homocysteine, vitamin B₁₂, citrulline, vitamin D, β -carotene, d-xylose absorption; (3) Prognostic Inflammatory and Nutritional Index (PINI); and (4) body composition and metabolism: body mass index (BMI), fat and lean body mass, resting energy expenditure (RRE), and respiratory quotient.

Results—Most patients had improvement in disease activity with infliximab. PINI decreased in all patients (-3.35 , $P = .04$). Plasma folate concentration significantly increased. There was an increase in BMI, fat mass, and lean body mass. The respiratory quotient increased in most patients. Changes in citrulline level and REE were inconsistent.

Conclusions—Crohn's disease patients have improvements in an index that measures both inflammation and nutrition (PINI) with infliximab therapy. Increases in plasma folate suggest improvement in enterocyte function and/or increased oral intake. The increase in respiratory quotient suggests decreased lipolysis and the lack of a starvation state. It was unclear whether weight gain was predominantly fat or lean muscle mass. These findings also support the use of PINI in Crohn's patients as an overall marker of inflammation and nutrition, and as a measure of response to infliximab therapy.

Keywords

Crohn's disease; infliximab; nutrition assessment; body composition

Crohn's disease (CD) is a chronic inflammatory disorder that can affect any part of the GI tract. Patients are often afflicted with various nutrition deficiencies at the time of diagnosis and throughout the course of their disease.¹ With progression, the disease causes serious malnutrition due to impaired absorption, nutrient loss, inadequate food intake, and drug-nutrient interactions. Patients are typically deficient in both macro- and micronutrients, such as depleted protein stores, hypoalbuminemia, water- and fat-soluble vitamin deficiencies (B₁₂, B₆, folate, vitamins D, E, A, K), and deficient mineral status (iron, zinc). Active disease is also associated with a starvation state characterized by an increase in resting energy expenditure (REE) and increase in lipolysis and proteolysis after a relatively brief fast.²⁻⁴

The severity of malnutrition tends to be correlated with the activity of CD; thus, controlling CD activity can often lead to improvements in nutrition status.⁵⁻⁷ Severe CD has typically been treated with anti-inflammatory and immunosuppressive drugs. Although there are obvious clinical and symptomatic improvements, nutrition deficiency is not always treated in patients taking these medications.⁶ In addition, many of the commonly prescribed medications for CD have been associated with loss of appetite, nausea, and vitamin deficiencies.^{2,8}

Recently, infliximab has been recognized as an effective treatment for patients with CD. Infliximab is an antibody to tumor necrosis factor α (TNF- α), a proinflammatory mediator in CD. High levels of TNF- α are associated with active CD and depletion of protein stores. Patients respond quickly to infliximab with average response and remission time of 1–1.5 weeks.⁹ Overall, patients receiving infliximab have lower rates of relapse, better Crohn's Disease Activity Index scores, and fewer CD-related hospitalizations compared with those who do not receive this drug.¹⁰

Although patients have obviously had significant improvement from infliximab therapy, an accurate measurement of improvement in nutrition status and the mechanism of improvement has not yet been established. This study addresses the issue of nutrition changes in Crohn's patients throughout the initial infliximab treatment. Because the pathophysiology of malnutrition is complex, we addressed several aspects in assessing nutrition status, including measurements of body composition, energy expenditure, and biochemical parameters for mucosal function with correlating disease activity indices.

Materials and Methods

Patients

Patients were recruited from the Department of Gastroenterology and Hepatology at the Cleveland Clinic. Participants were limited to patients with ileal or ileocolonic CD that had never received infliximab and had made the decision with their doctor prior to study enrollment to begin infliximab therapy. Patients with Crohn's colitis and no evidence of small bowel disease and patients with fistulizing CD as the primary indication for infliximab therapy were excluded. Disease location and behavior were previously defined by the patient's primary gastroenterologist per standard endoscopic and radiographic testing. Patients with a pacemaker, defibrillator, or any other implanted electrical device were also

excluded due to interference with the bioelectrical impedance measurements. Patients taking other medications were asked to maintain stable dosing throughout the first 6 weeks of the study unless the treating physician felt that a medication change was necessary. The study was approved by the Cleveland Clinic Institutional Review Board. Written informed consent was obtained from all participants.

Study Measures

Patients were assessed for disease activity, quality of life (QOL), and nutrition status within 4 hours prior to infliximab administration at weeks 0 and 6, and again at week 30. Evaluation points of 6 and 30 weeks were chosen to investigate short- and long-term changes in response to infliximab. All tests were completed at the General Clinic Research Center at the Cleveland Clinic.

Disease Activity

Disease activity was assessed by the Inflammatory Bowel Disease Questionnaire (IBDQ) and the Harvey Bradshaw index (HBI). The IBDQ is a 32-item questionnaire focusing on QOL in CD patients; scores > 170 are considered a good QOL.¹¹ The HBI is a concise form of assessing disease activity based on general well-being, abdominal pain, number of stools, and complications.¹² In addition, plasma C-reactive protein (CRP) level > 1.0 at the time of diagnosis was used as a marker of active disease.

Enterocyte Function/Nutrition Status

Enterocyte function was assessed by estimates of enterocyte mass, mucosal function, and vitamin and mineral levels. Levels of plasma citrulline served as an estimate of enterocyte mass. Although not studied extensively in CD, lower plasma citrulline concentration has been shown to be associated with villous atrophy and decreased functional absorptive bowel length.¹³⁻¹⁵ Citrulline was measured via automated ion-exchange chromatography in an amino acid panel. The following vitamins and minerals were measured by standard practice at the core laboratory of the Cleveland Clinic: red blood cell folate, plasma homocysteine, vitamin β_{12} , methylmalonic acid, vitamins 25-OH-vitamin D and β -carotene, serum iron, and total iron binding capacity (TIBC). In addition, patients underwent a standard 25 g d-xylose test for assessment of mucosal function. Measurements of d-xylose were restricted to preingestion and 2-hour postingestion blood measurements. The 2-hour interval between measurements is per Cleveland Clinic Laboratory Protocol.

Prognostic Inflammatory and Nutritional Index (PINI)

The PINI is an index of overall nutrition status and inflammation in critically ill patients.¹⁶ It requires serum measurements of α 1-acid glycoprotein (α 1-AGP), C-reactive protein (CRP), albumin (ALB), and prealbumin (PA). The PINI is calculated as $(\alpha 1\text{-AGP} \times \text{CRP})/(\text{ALB} \times \text{PA})$.

Body Composition

Body composition was assessed by 2 methods: bioelectric impedance analysis (BIA) and dual-energy X-ray absorptiometry (DXA) scan. Due to radiation exposure and cost of DXA

scans, this method of body composition assessment was utilized at the 0- and 30-week visits only. BIA poses no adverse effects for most patients, is less expensive, and is more accessible than DXA, and therefore, it was utilized at the 0-, 6-, and 30-week visits. We used a Quantum II analyzer (RJL Systems, Clinton Township, MI) for bioelectrical impedance analysis. The whole-body DXA scans were completed on the same Lunar Prodigy machine (GE Medical Systems, Madison, WI) by designated technicians.

Energy Expenditure

Patients' REEs were calculated by indirect calorimetry using a Jaeger Oxycon Pro (VIASYS Healthcare, Yorba Linda, CA). Measurements were taken in a supine position over 30 minutes after achieving steady state for at least 10 minutes. Patients were instructed to participate in minimal exercise for 24 hours before testing and to fast for at least 12 hours. The fasting respiratory quotient (RQ) was calculated by the equation VCO_2/VO_2 .

Treatment

Participants received standard infliximab therapy for CD consisting of an initial 5 mg/kg infusion followed by 5 mg/kg infusions 2, 6, and 8 weeks later. Maintenance therapy was every 8 weeks. Infusions were completed outside of the study.

Statistical Analysis

Participants served as their own controls with measurements at time 0 used as the reference for statistical purposes. A paired Student's *t* test was used to analyze normally distributed continuous variables. Inasmuch as this was a hypothesis-generating study, α error was set at .05 (2-sided), and we did not adjust for multiple comparisons.

Results

Disease Activity

Age, sex, and disease activity in the 7 patients are presented in Table 1. Of the 7 patients, 5 had active disease defined by CRP > 1.0, and 6 had active disease defined by HBI > 5. All patients had a poor disease-related QOL at time of enrollment as evidenced by IBDQ < 170. CRP improved significantly after 6 months of infliximab therapy (-0.120 , $P = .03$). The improvement in IBDQ and HBI after 6 months was not statistically significant ($+38$, $P = .06$, and -6.07 , $P = .22$, respectively).

Enterocyte Function

Levels of plasma folate increased after 6 months of infliximab therapy, with the greatest change in those with high CRP at time of enrollment ($+2.14$ ng/mL in all patients, $P = .04$ and $+3.30$ ng/mL in high CRP patients, $P = .0015$, respectively). Corresponding homocysteine levels decreased in 5 of 6 patients (-1.60 mmol/L, $P = .07$). Changes in serum levels of vitamin B₁₂, methylmalonic acid, citrulline, and the fat-soluble vitamins were inconsistent among patients. Changes in d-xylose absorption also varied among patients. Five of the 7 patients had an increase in iron levels and TIBC ($+21.43$ ug/dL, $P = .10$ and $+38.57$ ug/dL, $P = .15$, respectively; Table 2).

PINI

The PINI improved in all patients after 6 months of infliximab therapy (-3.35 , $P = .04$; Table 2, Figure 1). Those patients with active disease at time of enrollment had a greatest drop in PINI (-4.65 , $P = .03$) and experienced the greatest improvements in serum albumin and α 1-AGP levels ($+0.32$ g/dL, $P = .24$, and -113 mg/dL, $P = .06$, respectively).

Body Composition

There was little difference in body composition between the females and the male patient. There was 1 female outlier with an initial BMI of 33.2 kg/m². Overall, patients experienced an increase in BMI after 6 months of infliximab ($+2.21$ kg/m², $P = .03$; Table 3, Figure 2). There was a nonsignificant increase in lean muscle mass and body fat percentage as calculated by DXA ($+872.33$ g, $= 0.4$, and $+1.81\%$, $P = .1$). Overall, body fat percentage estimated per bioelectrical impedance increased ($+3.15\%$, $P = .08$).

REE

Changes in REE normalized per kg were variable among patients. For most patients, the RQ ratio increased slightly from 0.77 to 0.81 (all $+0.04$, $P = .6$, and high CRP $+0.05$, $P = .02$; Table 3).

Discussion

Treating both disease activity and nutrition deficiencies is an essential component for management of CD. Optimal care can affect the general well-being of patients as well as the mortality and morbidity of the disease.¹⁷ Identifying whether or not pharmacological therapy improves or worsens nutrition status is important for recognizing risk factors and side effects of current medications. Infliximab is currently an effective drug in improving disease activity, yet its effect on nutrition status has not been well studied.

Our study indicates that infliximab has positive effects on overall nutrition and inflammation as evidenced by a significant decrease in the PINI. Although some of this effect can be attributed to decreases in CRP, there were positive effects on serum levels of albumin and α 1-AGP in patients who had an elevated CRP level at time of enrollment. The PINI, based on a ratio of 2 acute phase proteins (CRP and α 1-AGP) to 2 visceral proteins (ALB and PA), was designed and tested to be a more sensitive tool for diagnosis and prognosis of stressed patients.¹⁶ Initial studies in acutely ill ICU and trauma patients have found the PINI served as an accurate predictor of clinical outcomes.^{18,19} In chronic renal failure patients receiving hemodialysis, increased PINI levels are associated with malnutrition and increased risk of comorbidities.²⁰ Cancer patients have significantly elevated PINI levels, and measurement of serial PINI levels has been proposed to assess overall nutrition and prognosis in this population.^{21,22} The PINI has not yet been validated in CD; however, its potential use is interesting. We selected this marker because of its reliability in other types of hospitalized patients and its ease of measurement. The PINI in our patients along with the other measures that we made support its use in CD. However, large-scale studies should be completed to evaluate whether this scoring system can be utilized routinely to assess inflammation and nutrition in CD.

The improvement in plasma folate with infliximab is important because approximately 30% of CD patients are afflicted with folate deficiency.²³ Low levels of plasma folate in inflammatory bowel disease (IBD) patients have been identified as risk factors for anemia, thromboembolic complications, and colon cancer.^{24,25} Given the high prevalence and consequences of folate deficiency in IBD, improvement in this vitamin level is of clinical importance. Folate deficiency in CD is attributed to decreased intake, increased intestinal losses, and interactions with medications such as sulfasalazine or methotrexate. In this study, the increase in red blood cell folate was likely due to both an improved intake and a decrease in intestinal losses. Most patients experienced an improvement in iron status. Although not significant, this trend is expected given the effects of infliximab on control of disease activity resulting in decreased intestinal bleeding and decreased inflammation. Improvements in iron absorption are unlikely given the limited involvement of the duodenum in CD. With such high rates of iron deficiency anemia in CD, this trend is of clinical importance.²⁶ Due to inconsistent changes among the other vitamins, little can be concluded regarding infliximab's effects on these micronutrients.

All patients experienced an increase in weight with infliximab treatment. This weight gain was due to an increase in both lean muscle mass and body fat. Ancel Keys's early starvation studies found a preferential deposition of fat mass during refeeding.²⁷ This has been confirmed in subsequent refeeding studies of cachectic patients.²⁸ Whether or not infliximab-induced weight gain follows a refeeding pattern of preferential fat mass deposition is unclear. TNF- α acts as a regulator of obesity by limiting adipocyte size and enlargement.²⁹ Interfering with TNF- α likely allows the body to decrease lipolysis and increase lipid deposition. A decrease in lipid breakdown is suggested by an overall increase in the RQ. Lower RQs have been found in patients with IBD compared with healthy controls and in patients with active IBD compared with nonactive IBD.^{2,30} The trend toward less lipid oxidation is consistent with other refeeding studies and suggests overall improved nutrition.³¹ Levels of TNF- α also have been associated with increased proteolysis.³² A recent study of children with IBD found that infliximab therapy decreases proteolysis and overall protein turnover.³³ Whether there truly is an increase in lean body mass (LBM) after infliximab therapy needs to be better clarified with larger studies. Because the majority of weight loss in CD is due to decreased intake, the increase in BMI was likely due to the increased nutrition intake from improved disease activity. Future studies will be needed to document this effect.

An increased REE is a potential contributor to malnutrition. It is thought that patients with CD experience an increase in REE that is associated with increased disease activity.⁴ Previous studies have shown that infusions of TNF- α are associated with increased energy expenditure.³⁴ A recent study in children with IBD found no significant changes in REE after 2 weeks of infliximab therapy.³³ The results from our study confirm no overall change in REE after 6 months of infliximab treatment. The effect of infliximab on controlling disease activity as well as its potential in altering both LBM and fat mass likely contributed to inconsistent changes in REE.³⁵

As this was a pilot study, our interpretations are limited by a small sample size and female preponderance. There was no parallel control group in the study. We did not include a

dietary assessment in our evaluation of changes in nutrition status. Future studies should assess how infliximab affects patients' intake and whether this can directly account for any of the changes found in this study.

Infliximab appears to have a role in improving nutrition status by decreasing overall inflammation leading to overall weight gain and decreased lipolysis. Our findings are important for furthering understanding of the role of infliximab as a treatment for severe Crohn's disease and its nutrition consequences. Further study in a larger group of patients would be of great interest.

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Figure 1.
Changes in Prognostic Inflammatory Nutritional Index (PINI) with infliximab therapy.

Figure 2.
Changes in body mass index (BMI) with infliximab therapy.

Table 1
Patient Characteristics and Crohn's Disease Activity Before and After Infliximab Treatment

Age	Mean: 41.1 (34-53)		
Gender	6 female, 1 male		
Mean	Week 0	Week 6	Month 6
IBDQ	111.29	149.71	149.29
HBI	12.93	8.00	8.00
CRP (mg/dL)	1.79	0.99	0.68*

CRP, C-reactive protein; HBI, Harvey Bradshaw Index; IBDQ, inflammatory bowel disease questionnaire.

* $P = .03$ vs week 0.

Table 2
Mean Enterocyte Function Before and After Infliximab Treatment

	Week 0	Week 6	Month 6	P Value*
RBC folate (ng/mL)	10.07	12.90	12.21	.05
Homocysteine (umol/L)	10.61	9.00	9.01	.07
Albumin (g/dL)	3.87	4.03	4.04	.24
Citrulline (umol/L)	30.43	33.00	29.00	.79
PINI	3.87	0.62	0.52	.04
Vitamin B ₁₂ (pg/mL)	499.29	436.14	526.57	.64
MMA (nmol/L)	130.96	114.67	137.08	.47
Vitamin D (ng/mL)	39.13	28.06	27.22	.40
β-carotene (ug/dL)	63.14	63.57	67.00	.64
Iron (ug/dL)	37.71	56.00	59.14	.10
TIBC (ug/dL)	323.14	363.14	361.71	.14

MMA, methylmalonic acid; PINI, Prognostic Inflammatory Nutritional Index; RBC, red blood cell; TIBC, total iron binding capacity.

* Month 6 vs week 0.

Table 3
Mean Body Composition and Metabolism Before and After Infliximab Treatment

	Week 0	Week 6	Month 6	P Value*
BMI (kg/m ²)	24.45	25.49	26.66	.03
% Body fat per BIA	32.43	33.14	35.58	.09
% Body fat per DXA	39.14		40.96	.10
LBM per DXA (kg)	39.16		40.03	.44
REE (kcal/kg)	22.20	23.83	21.56	.42
RQ	0.77	0.80	0.81	.07

BMI, body mass index; BIA, bioelectrical impedance; DXA, dual-energy X-ray absorptiometry; LBM, lean body mass; REE, resting energy expenditure; RQ, respiratory quotient.

* Month 6 vs week 0.