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Applied nutritional investigation

Specific carbohydrate diet for pediatric inflammatory bowel disease in clinical practice within an academic IBD center

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ABSTRACT

Objective: Despite dietary factors being implicated in the pathogenesis of inflammatory bowel disease (IBD), nutritional therapy, outside of exclusive enteral nutrition (EEN), has not had a defined role within the treatment paradigm of pediatric IBD within IBD centers. Based on emerging data, Seattle Children's Hospital IBD Center has developed an integrated dietary program incorporating the specific carbohydrate diet (SCD) into its treatment paradigm. This treatment paradigm uses the SCD as primary therapy as well as adjunctive therapy for the treatment of IBD. The aim of this study was to evaluate the potential effects of the SCD on clinical outcomes and laboratory studies of pediatric patients with Crohn's disease (CD) and ulcerative colitis (UC).
Methods: In this retrospective study, we reviewed the medical records of patients with IBD on SCD.
Results: We analyzed 26 children on the SCD: 20 with CD and 6 with UC. Duration of the dietary therapy ranged from 3 to 48 mo. In patients with active CD (Pediatric Crohn's Disease activity index [PCDAI] >10), PCDAI dropped from 32.8 ± 13.2 at baseline to 20.8 ± 16.6 by 4 ± 2 wk, and to 8.8 ± 8.5 by 6 mo. The mean Pediatric Ulcerative Colitis Activity Index for patients with active UC decreased from a baseline of 28.3 ± 10.3 to 20.0 ± 17.3 at 4 ± 2 wk, to 18.3 ± 31.7 at 6 mo.
Conclusion: This retrospective review provides evidence that the SCD can be integrated into a tertiary care center and may improve clinical and laboratory parameters for pediatric patients with nonstructuring, nonpenetrating CD as well as UC. Further prospective studies are needed to fully assess the safety and efficacy of the SCD in pediatric patients with IBD.

Introduction

Inflammatory bowel disease (IBD) is characterized by acute and chronic intestinal inflammation in the absence of a recognized etiology [1]. Although no specific causes have has been found, evidence from human and animal studies supports the hypothesis that patients with IBD have a dysfunction of the adaptive and innate immune system in response to the fecal microbiome [1]. Despite dietary exposures having been associated with the development of IBD and disease course [2,3], the primary therapies for IBD are medications that suppress the immune system or possess direct anti-inflammatory effects.

Nutritional therapy, outside of the use of exclusive enteral nutrition (EEN) for Crohn's disease (CD), has not had an established role within pediatric IBD [4–6]. To date, nutritional therapy aimed at modifying disease activity has primarily referred to formula-based enteral nutrition. EEN has been shown to alleviate clinical symptoms, improve an individual's nutritional status, and improve abnormal laboratory parameters associated with active inflammation. In children with CD, EEN has been effective at inducing clinical remission, and superior at achieving mucosal healing when compared with steroids [7,8].

Our group, as well as others, has demonstrated that dietary intervention including the specific carbohydrate diet (SCD) has efficacy in CD [9,10]. The SCD that was used initially to treat celiac disease in the mid 20th century, and was popularized in the 1990s, removes grains including wheat, barley, corn, and rice;

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and uses nut flours such as almond and coconut flours to make breads and other baked goods. Additionally, added sugar is limited to honey. The diet also restricts most milk products except for fully fermented yogurt. Although the mechanism of action for the SCD is not known, it is hypothesized that the diet decreases intestinal inflammation by changing the fecal microbiome from a proinflammatory state to noninflammatory state [11]. Since our initial report of efficacy of the SCD, further studies showing efficacy of dietary intervention have been reported [10, 12]. Based on emerging data, Seattle Children's Hospital IBD Center developed an integrated dietary program incorporating the SCD into its treatment paradigm. This treatment paradigm uses the SCD as primary therapy for IBD as well as adjunctive therapy when partial response to medication occurs. This retrospective study reviews the medical records of patients with IBD on the SCD since the initiation of the SCD protocol. The aim of this study was to evaluate the potential effects of the SCD on clinical outcomes and laboratory studies of pediatric patients with CD and ulcerative colitis (UC).

Materials and methods

We initiated a retrospective chart review of children with CD and UC seen at Seattle Children's Hospital from December 2012 to December 2014 who had been on SCD therapy. The protocol was approved by the Seattle Children's Hospital Institutional Review Board (IRB study #15309). Criteria for inclusion in the analysis were patients who had trialed the specific carbohydrate diet as part of their treatment for IBD. All data was extracted from electronic medical records. The diagnosis of CD or UC was based on conventional criteria, including clinical, radiologic, endoscopic, and histologic findings. Assessment of disease activity was evaluated with the abbreviated Pediatric Crohn's Disease activity index (PCDAI)/Pediatric Ulcerative Colitis Activity Index (PUCAI); remission was defined as a PCDAI or PUCAI score of $\leq$10. Mild to moderate active CD was defined as PCDAI between 10 and 30 and mild to moderate active UC was defined as PUCAI between 10 and 65. Laboratory data used to assess IBD-related inflammatory activity included erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), albumin, and fecal calprotectin; anemia was assess by hematocrit. Nutritional and growth status was evaluated with anthropometrics including body mass index (BMI) and height velocities. Patient information was obtained from clinic visits before initiating the SCD up to 12 to 14 mo post-diet initiation.

Statistical methods

All demographic, clinical, and laboratory variables were summarized using frequencies and percentages for categorical variables and mean, median, range, and interquartile range (IQR) for continuous variables, as appropriate. Two-sample *t* tests were used to evaluate differences in age and duration variables between cases and controls. Linear mixed-effects models [13] were used to evaluate differences between cases and controls in variables such as the PCDAI and PUCAI, which were measured over time, using a within-patient spatial correlation matrix to account for the correlation of unequally spaced repeated measurements. These analyses provided tests of linear trends within the case and control groups, as well as comparisons between the trends. All significance testing was conducted at the $\alpha = 0.05$ level. SAS 9.4 (SAS Institute Inc., Cary, NC, USA) were used for all analyses. All figures were constructed using R 3.0.3 [14].

Results

Fifty pediatric patients with IBD were identified for this review. Patients previously reported in the medical literature were excluded from the analysis (n = 7) as well as those patients enrolled in our current prospective study (n = 7; Clinical trial.gov number 14956). In all, 36 patients met inclusion criteria. Of these, 26 were able to remain on the SCD >2 wk. Ten individuals were not able to maintain on the SCD and began standard medical therapy. These patients acted as controls for this study.

SCD patients and disease characteristics

Twenty-six patients were able to maintain on the SCD. Of these, 20 patients were diagnosed with CD. Six were diagnosed with UC . Thirteen patients were boys and 13 were girls. The mean age of patients at initiation of diet therapy was 10.9 y, with a range of 1.5 to 19 y. Twenty-three patients were white, non-Hispanic ethnicity; one declared Asian race, non-Hispanic ethnicity; two self-identified as both white and Asian (Table 1).

For the patients with CD, 12 of the 20 had upper tract disease located within the stomach and duodenum. Five had ileocolonic disease and one had isolated colonic disease. One patient had a limited colonoscopy and disease extent could not be defined. No patient had penetrating or stricturing disease. For patients with UC, one had pancolitis (disease proximal to hepatic flexure), four had left-sided disease (distal to splenic flexure), and one had extensive disease (hepatic flexure, distally) [15].

Fifteen patients were on concurrent medication/therapy with SCD therapy including mesalamine (n = 5), methotrexate (n = 5), azathioprine (n = 2), biological therapies (n = 6), and enteral nutrition (n = 3), as well as helminthic therapy (n = 1). The remaining patients (n = 11) were not on IBD-related medications.

Patients' primary reasons for initiating the SCD therapy included avoidance of medication (n = 17), partial improvement to medication (n = 5), no improvement to medication (n = 3), side effect from medications (n = 1).

Duration of the SCD therapy ranged from 3 to 48 mo with a mean of 9.6 $\pm$ 10.1 mo. Two patients started the SCD soon after the diagnosis of IBD; they received no other induction therapy. Thirteen patients had induction therapy with medication and elected to use the SCD for maintenance therapy in conjunction with medical therapy. Three patients were on standard medical therapy to induce remission and used the SCD as sole maintenance therapy. Two of these patients received an initial course of prednisone and mesalamine treatment immediately after diagnosis as induction therapy and were later maintained exclusively on SCD. The other patient was clinically feeling well on mesalamine and azathioprine therapy but had concerns for poor weight gain and continued elevated inflammatory markers. This patient therefore began SCD therapy. Two additional patients did not fully respond to standard medical induction therapy and elected to use the SCD as induction and maintenance therapy. Six patients received exclusive enteral nutritional (EEN) therapy for 2 mo before transitioning to the SCD.

Control group and disease characteristics

Ten pediatric patients with IBD acted as controls, seven with CD and three with UC. These patients were not able to maintain the SCD and transitioned to standard medical therapy. Five patients were girls. The mean age of the control group was 14.4 $\pm$ 3.1 y with a range of 10 to 19 y of age. For patients with CD, three had ileocolonic disease, two had both ileal and upper tract disease, two had disease limited to the ileum. For patients with UC, two had pancolitis, and one had ulcerative proctitis. All 10 patients were on medication therapy including mesalamine (n = 2), antibiotics (n = 1), steroid therapies (n = 4), immunomodulators (n = 4), and biologic therapies (n = 7); one patient was on enteral nutrition.

Crohn's disease: symptoms and laboratory evaluations

Patients initially presented with CD with a variety of symptoms: abdominal pain (n = 7), weight loss (n = 8), blood per

Table 1
Patient demographic characteristics and clinical classification on specific carbohydrate diet

ID	Sex	Age (y)	CD duration before diet (y)	Diet duration (mo)	Paris classification*	Medications and nutritional therapy (while on diet)	SCD diet type† (food items added without symptom)
Crohn's disease							
1	Female	10	3	4	A1 a L3 L4 a B1 P G1		SCD (strict)
2	Female	11	3	20	A1 a L3 L4 a B1 G0		SCD (like)–rice, potatoes
3	Male	14	1	10	A1 b L3 B1 G1	Infliximab, mesalamine	SCD (like)–nos
4	Female	10	1	48	A1 a L3 B1 P G1		SCD (strict)
5	Male	19	3	12	A1 b L3 B1 G1	EN‡	SCD (strict)
6	Male	7	1	9	A1 a L2 B1 G1	Infliximab, methotrexate, EN	SCD (like)–simple starch; potatoes, oatmeal, cheese, milk
7	Female	13	1	6	A1 b L3 B1 P G1		SCD (strict)
8	Male	12	<1	4	A1 b L2 L3 B1 P G0		SCD (like)–processed Greek yogurt, ice cream with additive gums
9	Male	13	3	8	A1 a L2 L4 a B1 P G0	Infliximab, methotrexate, mesalamine	SCD (strict)
10	Male	1.5	<1	4	A1 a L B1 G1§	EN	SCD (like)–nos
11	Female	6	4	5	A1 a L3 L4 a B1 G0	Mesalamine	SCD (strict)
12	Male	4	3	10	A1 a L2 B1 G1	EN	SCD (like)–occasional wheat products, kefir yogurt, dairy butter
13	Male	6	1	3	A1 a L2 L4 a B1 G0	Azathioprine, mesalamine	SCD (like)–nos
14	Female	16	6	5	A1 a L3 B1 G0	Ustekinumab, EN	SCD (like)–nos
15	Male	10	1	8	A1 a L2 B1 G1	mesalamine	SCD (strict)
16	Male	8	1	31	A1 a L2 L4 a B1 G1		SCD (strict)
17	Male	15	<1	3	A1 b L3 L4 a B1 P G0		SCD (strict)
18	Female	9	1	6	A1 a L3 L4 a B3 P G0		SCD (like)–rice
19	Male	15	1	3	A1 b L2 L4 a B1 G1	Infliximab, methotrexate	SCD (like)–nos
20	Male	10	2	3	A1 a L3 L4 a B1 G0	Methotrexate	SCD (strict)
Ulcerative colitis							
21	Female	11	<1	3	A1 b E3 S0	Steroid retention enemas	SCD (strict)
22	Female	14	<1	20	A1 b E2 S1	6-MP	SCD (strict)
23	Female	16	3	5	A1 b E2 S1	Azathioprine	SCD (like)–nos
24	Female	16	5	4	A1 b E4 S1	EN	SCD (like)–nos
25	Male	10	1	8	A1 b E3 S1	Curcumin	SCD (like)–Paleo diet
26	Female	9	2	8	A1 a E2 S1	Whipworm therapy, mesalamine	SCD (like)–rice, gluten-free

6-MP, 6- mercaptopurine; CD, Crohn's disease; EN, enteral nutrition; nos, not otherwise specified; SCD, specific carbohydrate diet; UC, ulcerative colitis

* See reference [13].

† SCD is defined as "strict" if no "illegal foods" have been added to the diet based on criteria previously referenced [11]. "SCD-like" diet is defined as diet by way of adding one of more "illegal foods."

‡ EN is defined as enteral nutrition via supplemental formula.

§ Patient 10 had incomplete visualization of mucosal inflammation on diagnostic colonoscopy and endoscopy.

rectum (n = 8), chronic diarrhea (n = 10), and fatigue (n = 3). Table 2 shows an abbreviated PCDAI for patients while on dietary therapy [16,17]. The mean abbreviated PCDAI dropped from 14.5 ± 16.4 before SCD to 8.5 ± 13.4 at 4 ± 2 wk and to 3.1 ± 5.1 by 6 mo (Table 2). For patients with CD initiating the diet while having an active flare (n = 9; PCDAI >10), PCDAI dropped from 32.8 ± 13.2 before SCD to 20.8 ± 16.6 at 4 ± 2 wk, and to 8.8 ± 8.5 by 6 mo. The nine patients with elevated PCDAI (>10) had a decrease in PCDAI once beginning the SCD; of these seven were able to achieve clinical remission (PCDAI ≤10). Eleven of the 20 CD patients began the diet in remission. Ten maintained remission on the diet. Of those 20 patients who initiated the SCD, 13 have continued on the SCD (Fig. 1). Two patients were able to remove adjunctive medication (i.e., methotrexate). Seven of the 20 patients discontinued the SCD; 1 patient clinically flared, 2 did not see improvement on the diet, and 4 discontinued the diet secondary to noncompliance/difficulty maintaining on the diet.

CRP normalized in 10 of 14 children with CD that had elevated levels before diet therapy (Fig. 2). Five of nine CD patients with abnormal sedimentation rates before initiation of the diet had normalization of these rates. CRP, sedimentation rate, and albumin improved on the diet for the majority of CD patients with the mean CRP going from 1.8 ± 1.0 mg/dL to 1.3 ± 1.1 mg/dL at 4 ± 2 wk, and to 0.9 ± 0.2 mg/dL by 6 mo. The mean sedimentation rate went from 19 ± 15.9 mm/h to 12.2 ± 7.6 mm/h at 4 ± 2 wk and to 9.5 ± 5.2 mm/h by 6 mo. The mean albumin went from 4.1 ± 0.5 g/dL to 4.3 ± 0.3 g/dL at 4 ± 2 wk, and to 4.3 ± 0.3 g/dL by 6 mo. Thirteen of the 20 patients had stool calprotectin checked. Calprotectin improved on the diet for 6 of 13 CD patients with the mean calprotectin level going from 685 ± 205 mcg/g to 212 ± 235 mcg/g at 4 ± 2 wk, and to 504 ± 540 mcg/g by 6 mo (Fig. 2).

The CD control group presented with multiple symptoms at diagnosis including abdominal pain (n = 1), diarrhea (n = 4), rectal bleeding (n = 3), anemia (n = 1), weight loss (n = 3), and perianal disease (n = 1). All patients initiated standard medical therapy. The mean abbreviated PCDAI dropped from 15 ± 11.9 at baseline to 9.2 ± 17.7 at 6 mo to 7.5 ± 10 at 12 mo while on medication maintenance therapy. Three patients began the study period in clinical remission but with elevated inflammatory markers. Two of these patients flared within the year and transitioned to biologic therapy. Four patients began the study with active disease. Of these, three were able to achieve and maintain remission on standard medication therapies. CRP normalized in three of six children with CD. One of two patients with abnormal ESR achieved normalization. Two patients with abnormal fecal calprotectin levels displayed improvement after medical treatment. Mean CRP improved from 4.5 ± 4.2 mg/dL to 1.9 ± 2.4 mg/dL at 6 mo on standard medication therapy. The mean ESR went from 33.5 ± 6.4 mm/h to 16.9 ± 12.4 mm/h at 6 mo to

Table 2
Means of measured parameters

Crohn's disease										
Parameter	Before diet		2–6 wk		4–6 mo		7–11 mo		12 mo	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PCDAI (all)	14.5	16.4	8.5	13.4	3.1	5.1	7.5	6.5	0	
PCDAI (diet initiated during active disease)	32.8	13.2	20.8	16.6	8.8	8.5	10	8.7	0	
ESR (mm/h)	19	15.9	12.2	7.6	9.5	5.2	11.8	10.1	14	16.1
CRP (mg/dL)	1.8	1.0	1.3	1.1	0.9	0.2	1.3	1.6	0.9	0.3
Albumin (mg/dL)	4.1	0.5	4.3	0.3	4.3	0.3	4.2	0.5	4.4	0.5
Hematocrit (%)	35.6	2.9	37.3	3.5	39.3	2.4	38.3	3.6	39.3	3.3
Calprotectin (mcg/g)	685	205.5	212.6	235	504	540.6				
Vitamin D, 25-hydroxy	31.1	4.7	30.7	10.6	37.5	26.2	34.3	13.6	24.5	3.5
BMI	17.3	2.3	17.9	2.3	16.7	2.9	16.9	3.12	18.3	4
Ulcerative colitis										
PUCAI (all)	20	11.4	12.5	13.7	12	24.1	10	–	0	–
PUCAI (diet initiated during active disease)	28.3	10.3	20	17.3	18.3	31.7	10	–	0	–
ESR (mm/h)	15.6	7.0	11.4	6.9	8.5	3.5	7	–	7	–
CRP (mg/dL)	1.0	0.5	0.7	0.3	0.9	0.3	0.8	–	0.8	–
Albumin (mg/dL)	4.2	0.4	4.5	0.2	4.4	0.3			4.1	–
Hematocrit (%)	35.1	2.6	38.9	3.2	37.5	3.0	35.5	–	38.5	–
Vitamin D, 25-hydroxy	25.5	3.5	28	0						
BMI	17.2	2.1	17.6	1.6	18.0	1.9	18.9	–	20.1	–

BMI, body mass index; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; PCDAI, Pediatric Crohn's Disease Activity Index; PUCAI, Pediatric Ulcerative Colitis Activity Index

12.3 ± 11 mm/h at 11 mo. Calprotectin improved on diet from 1431 ± 366 mcg/g at diet initiation to 969.5 ± 540.9 mcg/g at 4 ± 2 wk of standard therapies.

Anthropometric data for CD

Weight and height velocities were within normal range for all children with CD on SCD except for five who experienced some weight loss with negative weight velocity (Fig. 3). BMI for patients with CD increased from a mean of 17.3 ± 2.3 kg/m^2 to 18.3 ± 4 kg/m^2 by 12 mo of diet therapy (Table 2). Three of 20 CD patients began diet therapy with BMI below normal. Of these three patients, two experienced increase in BMI; two had complete normalization of BMI by 6 mo. Weight and height velocities were within normal range for all but two control patients with CD. BMI for the CD control group increased from a mean of 18.58 ± 3.2 kg/m^2 to 23.3 ± 2.6 kg/m^2 by 12 mo of therapy.

UC: symptoms and laboratory evaluations

Patients with UC presented with symptoms that included abdominal pain (n = 4), chronic diarrhea (n = 2), blood per rectum (n = 4), and weight loss (n = 2). Table 2 shows a PUCAI, a validated measure of disease activity, for patients while on dietary therapy [18]. The mean PUCAI decreased from before SCD of 20 ± 11.4 to 12.5 ± 13.7 at 4 ± 2 wk (Table 2). The mean PUCAI for patients with active UC decreased from a baseline of 28.3 ± 10.3 to 20 ± 17.3 at 4 ± 2 wk, to 18.3 ± 31.7 at 6 mo. Four patients initiated the diet with active disease (PUCAI >10), two of these patients went into clinical remission based on PUCAI. Two initiated the diet in remission and maintained remission thereafter. Of the six UC patients who initiated the SCD, three have maintained on the diet (Fig. 1). One patient, who had complete resolution of gastrointestinal symptoms, had continued joint pains and was transitioned to methotrexate. Two other patients with active disease did not respond to the diet.

Laboratory values for UC patients on dietary therapy improved or remained normal (Fig. 2).

UC control group patients presented with symptoms including abdominal pain (n = 1), and rectal bleeding (n = 3). The mean PUCAI decreased from 18.3 ± 12.6 at initiation of study to 10 ± 14.1 by 6 mo of therapy. Two of the three UC control patients initiated diet therapy during active disease. One UC control maintained remission; while one elected colectomy for ongoing symptoms and third had ongoing symptoms not resolved with medical therapy.

Anthropometric data for UC

Weight and height velocities where within normal range for all children with UC at initiation of SCD and throughout duration of SCD (Fig. 3). BMI for patients with UC increased from a mean of 17.2 ± 2.1 kg/m^2 to 18.0 ± 1.9 kg/m^2 by 6 mo of diet therapy (Table 2). Two patients form the UC control group had low weight and height velocities but otherwise normal anthropometrics. BMI for control patients with UC increased from a mean of 24.8 ± 7.5 kg/m^2 at diet initiation to 24.5 ± 6.8 kg/m^2 at the end of the study period on standard medication therapies.

Comparative analysis

A comparative analysis of the patients on the SCD versus controls, revealed significant improvement in PCDAI, CRP, and calprotectin over time for both groups ($P = 0.03$, 0.03, and 0.03, respectively). No difference was seen in the time trend between SCD cases and controls in regards to PCDAI; although there was a constant shift in the trend over time between SCD cases and controls in regard to CRP and calprotectin ($P = 0.002$ and 0.006, respectively). No significant difference was seen between SCD cases and controls in regard to PUCAI; however, the number of UC patients in both the SCD and control groups was small. Also, there was a similar constant shift seen for BMI, height, and weight between SCD and controls ($P = 0.01$, 0.03, and 0.009,

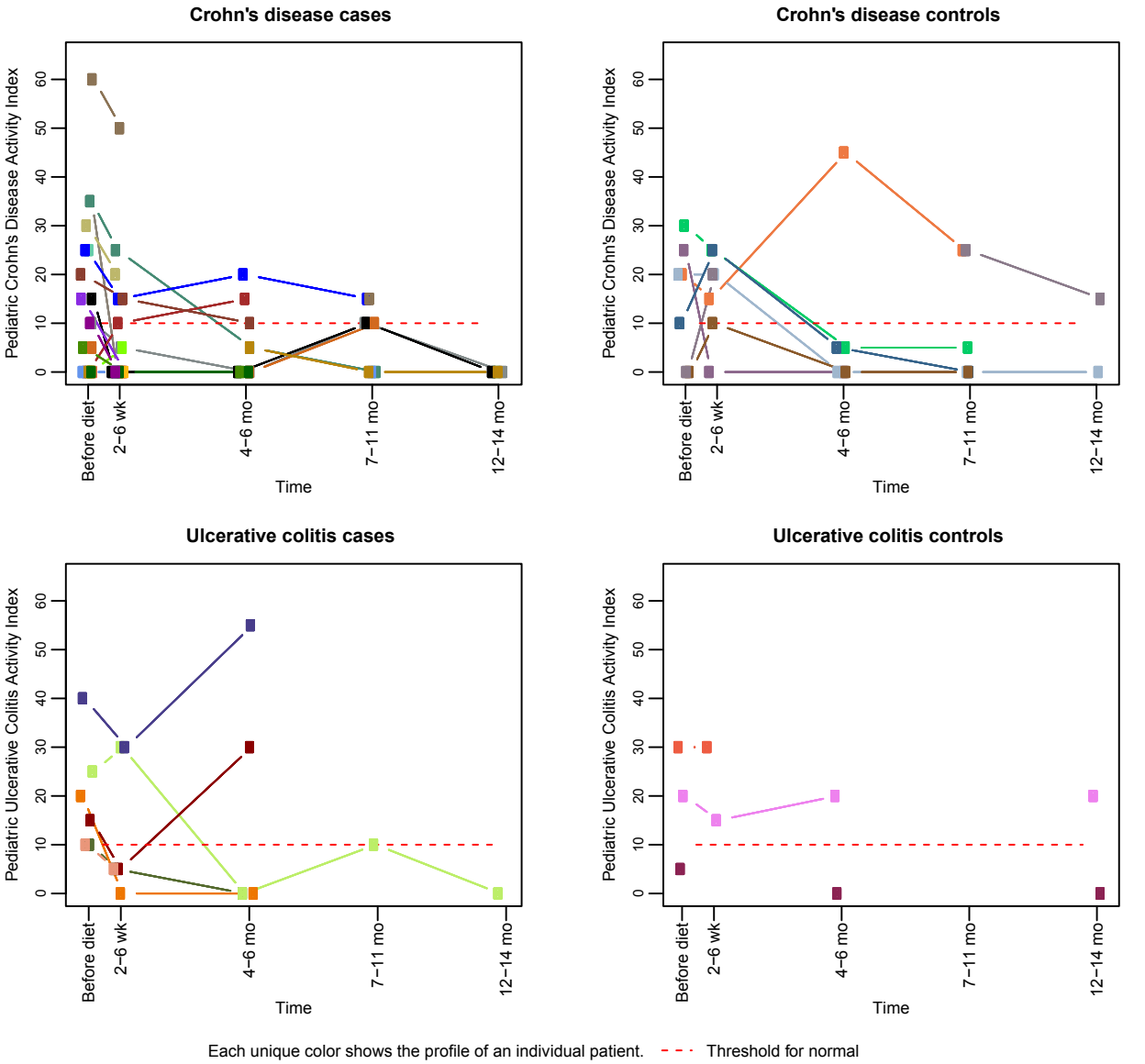

Fig. 1. Validated disease activity index score for iBD patient's on dietary therapy and controls.

respectively). These are all in the direction of lower for SCD patients versus controls.

Discussion

Nutritional therapy in the form of EEN is a fundamental component of patient care in pediatric IBD. Recent studies using formula-based therapies have amended standard EEN with the addition of dietary foods [12]. It has been shown that diet itself may be able to modify both inflammatory processes and symptoms in CD [9,10]. Dietary therapy in the form of SCD has been integrated into our clinical practice for those patients and patient families invested in trying a monitored nutrition-based therapy with clearly outlined goals including clinical remission and growth improvement. Within our practice, the SCD has been tried as sole therapy and as combination therapy with medications for both patients with UC and CD. Our data suggest that the SCD can be successfully integrated into an academic practice. Our findings build on our initial study showing both clinical and laboratory improvement for pediatric patients with IBD [9].

The results of this retrospective study show that within a small sample size of pediatric patients, the SCD has a positive effect on both symptoms and inflammatory markers. Although patient backgrounds, disease phenotypes, medication history, and reasons for electing the SCD as therapy were diverse, almost all patients who were able to maintain the diet demonstrated clinical improvement on the SCD. Of the 26 patients who maintained on the SCD, 12 had experienced both clinical and inflammatory marker improvements. Successful maintenance of remission with the SCD allowed some patients to discontinue medications and maintain disease control on the SCD alone. For those who were successfully able to reach clinical remission with the diet, weight velocity and height velocity stayed within standard range for patient age.

Despite the clinical and laboratory improvement seen with the SCD, the diet was difficult to maintain for many patients. Addition of selected foods was necessary to provide some

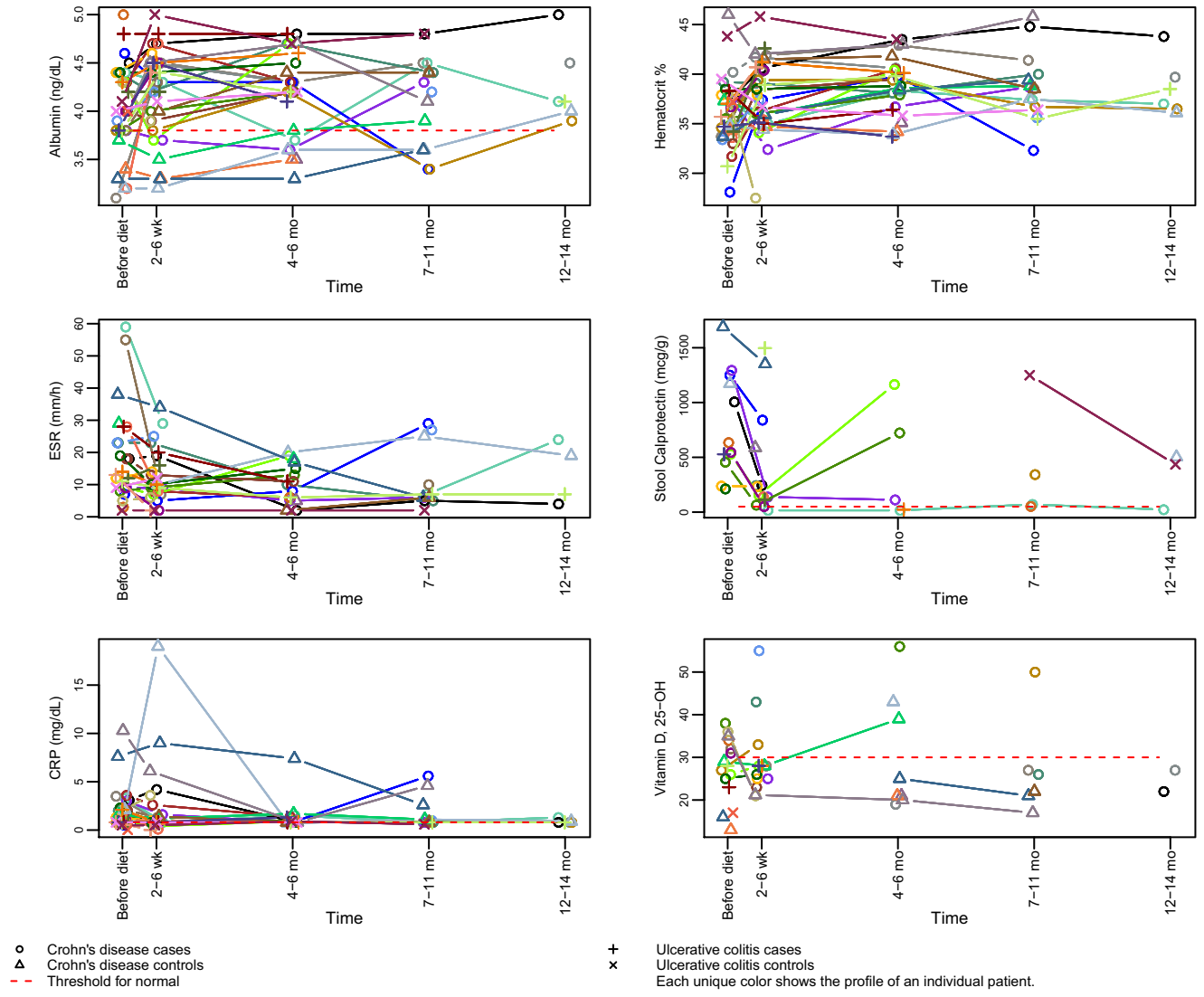

Fig. 2. Laboratory studies for IBD patients on dietary therapy and controls.

patients more dietary options so that they would maintain the diet. Liberalization of the diet included rice, oatmeal, potatoes, and cocoa powder. Adding new foods runs the potential risk for worsening disease activity; therefore when adding a new food baseline labs including stool calprotectin we requested and repeated 1 mo after addition of the food. If symptoms appeared or increase stool calprotectin was noted, that food was removed.

The SCD is not without potential adverse effects. For those who continued on the diet, nine patients experienced weight loss. To assure early identification of nutritional deficiencies, the SCD program at Seattle Children's Hospital includes close follow-up with both the physician and a dietitian educated in the SCD. Additionally, ensuring a "normal" quality of life on a rigorous diet such as the SCD may prove challenging for many patients and their families with potential isolation from one's peers. We address this issue by making sure that the patient is part of the decision-making process. This is also discussed during follow-up visits. Furthermore, the diet may potentially become a stressor between patient and parent. To avoid this situation in our clinical practice, the children themselves are a part of the decision-making process and can elect to halt diet therapy at anytime.

With the fecal microbiome implicated in the pathogenesis of IBD and with diet having a major effect on the composition of the fecal microbiome, diet as part of a therapeutic approach to patient care has a scientific basis. Animal studies have linked diet to the development on IBD in genetically predisposed animals. A Western diet rich in fats and simple sugars was compared with regular chow in CEABAC10 mice. The diet was shown to induce dysbiosis with increase in *Escherichia coli*, as well as alter host barrier function favoring adherent invasive *E. coli* [19]. Additionally, common dietary emulsifiers, carboxymethylcellulose and polysorbate-80, have been shown in mouse models to induce low-grade inflammation and metabolic syndrome in wildtype mice and promote a robust colitis in genetically predisposed mice. The emulsifiers were noted to change the composition of the microbiota increasing the overall inflammatory potential of the fecal microbiota. Additionally, these emulsifiers increased mucolytic bacteria causing erosion of the mucus layer and shortening the distance between fecal microbiota and intestinal epithelial cells by >50% [20]. Maltodextrin, another common food emulsifier has shown similar affects in animal models [21,22]. Conversely, potential positive effects of the SCD

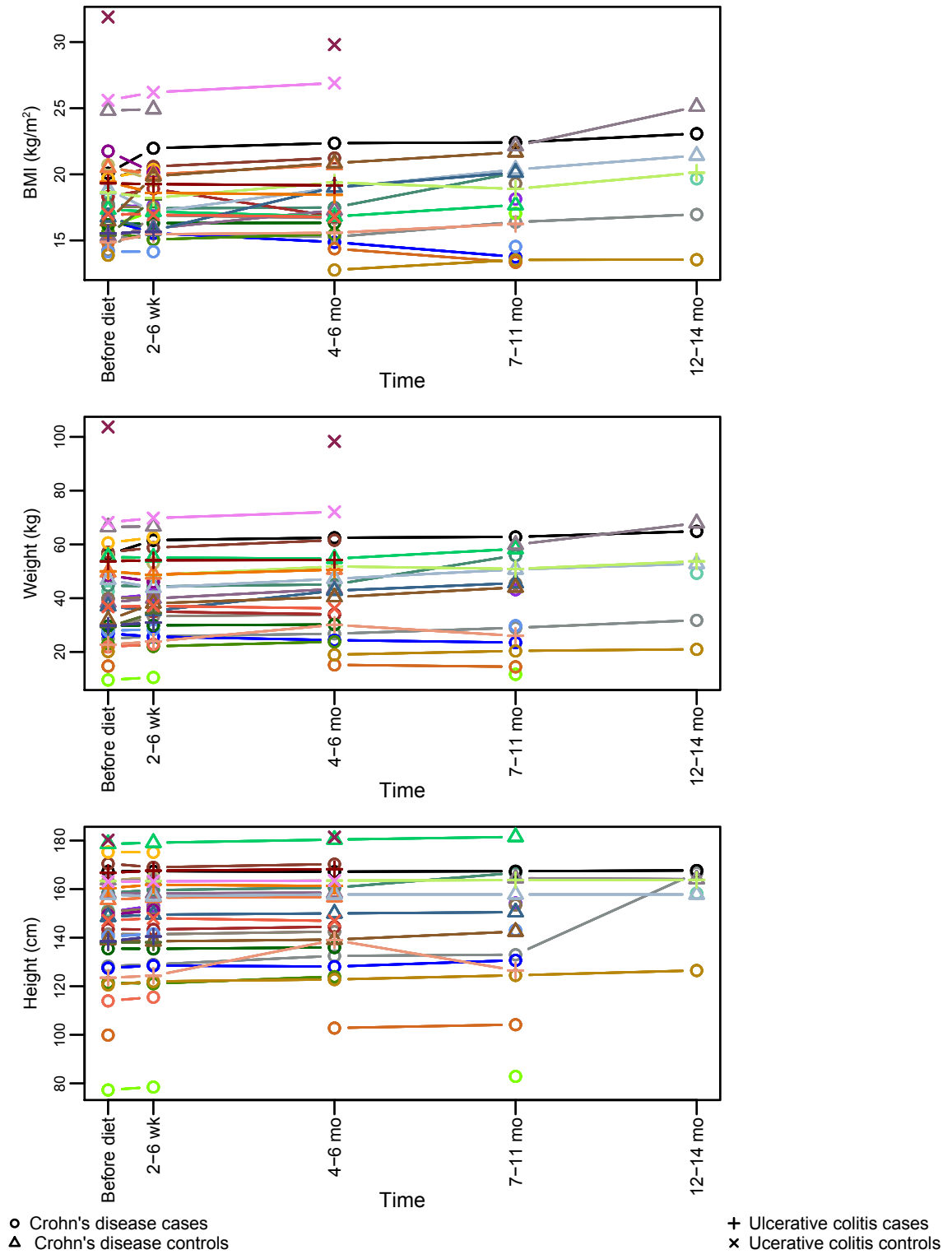

Fig. 3. Anthropometric measures in IBD patients on dietary therapy and controls.

on the fecal microbiome were seen in individuals with stable CD. Enrichment of microbial diversity was seen in patients on SCD as compared to a low residual diet [23]. Given the constructs of the SCD in which high-fat, high-sugar foods and most food additives (i.e., food emulsifiers) are removed, a potential mechanism of action exists for the clinical benefits we have seen.

There are limitations to the present study, including its retrospective and descriptive nature, small sample size, variation

of dietary monitoring, and patient compliance to the diet, as well as medications for induction and maintenance therapy and their potential influences on SCD efficacy. Additionally, the disease extent for the UC patients on the SCD is not representative of the usual pediatric UC population and reflects milder disease than what is usually seen. Despite these limitations, this study suggests that the SCD may be a possible therapeutic option for pediatric patients with CD and UC and can be successfully integrated into an academic IBD center. By partnering with families and being able to provide a structured integrated dietary program, our IBD center has been able to offer individualized care for patients and families who may have otherwise sought care outside of an IBD center or pediatric gastroenterology practice. This also allows us to address the potential conflicting dietary data families find on the Internet [24]. Further prospective studies are required to fully assess the safety and efficacy of any specific diet in patients with pediatric IBD.

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