Dietary Counseling Versus Dietary Supplements for Malnutrition in Chronic Pancreatitis: A Randomized Controlled Trial

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Background & Aims: Up to 50% of patients with chronic pancreatitis (CP) are malnourished. There are limited data on the role of dietary intervention in improving the nutritional status of such patients. The aim was to compare the efficacy of medium chain triglyceride (MCT)–enriched commercial dietary supplements with dietary counseling for homemade food in the management of malnutrition in patients with CP. Methods: In a randomized controlled trial, consecutive undernourished patients with CP (body mass index [BMI] < 18.5 kg/m²) at a tertiary care hospital were randomized to receive either dietary counseling for regular homemade food or commercial MCT-enriched dietary supplements for a period of 3 months to compensate for the dietary calorie deficit. All patients received standard management for CP including pancreatic enzyme supplements. Primary outcome measure was improvement in BMI. Results: Sixty malnourished patients with CP were randomized to counseling group (n = 29; mean age, 32 ± 10 years; male, 83%) and supplementation group (n = 31; mean age, 28 ± 10 years; male, 84%). BMI increased in both the counseling group and supplementation group (17.2 ± 1.7 vs 18.1 ± 1.8 kg/m², P = .001; 16.7 ± 1.6 vs 18.2 ± 1.6 kg/m², P = .001). There were similar improvements in triceps skinfold thickness, dietary intake, fecal fat, and pain score during a period of 3 months in both groups. There was, however, no significant difference between the counseling and supplementation groups with regard to any of the outcome measures. Conclusions: Dietary counseling for a balanced homemade diet is as good as commercial food supplements in improving malnutrition in patients with CP.

Chronic pancreatitis (CP) is characterized by pancreatic inflammation and fibrosis, eventually leading to destruction of pancreatic parenchyma and loss of exocrine and endocrine function. The most common cause of CP is alcohol in 40%–80% of cases, but it is idiopathic in 20%–60% of cases. Malnutrition is a common feature of CP, particularly in patients with alcoholic and idiopathic tropical CP. Although malnutrition has been thought of as a cause of or contributory factor in the pathogenesis of CP, others and we have shown that malnutrition is an effect and not a cause of CP. The etiology of malnutrition in these patients is multifactorial. Malnutrition caused by decreased pancreatic exocrine secretion and inadequate bicarbonate delivery to the duodenum leading to secondary inactivation of enzymes and bile acids by gastric acid is an important cause for malnutrition. Abdominal pain, nausea, vomiting, and postprandial satiety contribute by limiting dietary intake. Self-imposed dietary restriction caused by the fear of inducing pain also contributes to poor nutrition. Physicians also, as a general habit, advise patients with CP to reduce their fat intake to a minimum. In a study, we found that patients with CP had a selective dietary fat restriction due to food fads in spite of being adequately supplemented with pancreatic lipase and other enzymes and analgesics. Patients with alcoholic CP are malnourished as a result of continued alcohol intake as an important cause of undernutrition. Development of CP-related complications like diabetes, pancreatic pseudocyst, and pancreatic cancer also lead to nutritional decline. Hypermetabolic state with increased resting energy expenditure is another cause of malnutrition in 30%–50% of patients with CP. The degree of undernutrition has a negative impact on the outcome of these patients. Micronutrient deficiency might also contribute to decreased antioxidant capacity and increased oxidative stress in these patients.

Although a lot of emphasis has been laid on treating abdominal pain by way of analgesics, pancreatic enzyme supplements, endoscopic therapy, and surgery, not many studies have looked at strategies for improving nutrition in these patients beyond pancreatic enzyme supplementation. Recently, commercially available dietary supplements containing hydrolyzed oligopeptides and medium chain triglycerides (MCT) have been regarded as useful for improving nutrition in patients with CP. These are readily digestible, well-tolerated preparations. Moreover, patients might be more compliant, thinking it is of medicinal value. Recent studies have shown that the presence of MCT in the commercially available food supplements results in only a minimal stimulation of postprandial CCK release and exocrine pancreatic secretion, which might decrease abdominal pain in patients with CP. No study has explored the efficacy of these supplements as compared with simple dietary counseling with homemade food in improving the nutritional status of malnourished patients with CP.

We conducted a randomized controlled trial (RCT) to compare the efficacy of MCT-enriched commercially available food supplements with dietary counseling for regular homemade food for the management of malnutrition in patients with CP.
Methods

We conducted an RCT in our tertiary care academic center. Consecutive patients with CP attending the pancreas clinic at our center were included in the study during the period starting August 2000–July 2003. The diagnosis of CP was suspected on the basis of suggestive clinical features, ie, recurrent or chronic abdominal pain and/or presence of diabetes and/or steatorrhea. The diagnosis was confirmed if there was evidence of pancreatic calcification and/or ductal changes in the form of irregularity, dilation, and/or stricture of pancreatic duct on imaging studies that included ultrasonography and/or computed tomography (CT) of the abdomen and/or endoscopic retrograde cholangiopancreatography (ERCP)/magnetic resonance cholangiopancreatography (MRCP).12 Patients were assessed for their nutritional status. Patients with malnutrition formed the study group. Patients were considered malnourished if their body mass index (BMI) was less than 18.5 kg/m², or if they had lost significant weight (defined as recent loss of >10% of their usual body weight within the last 6 months) as a result of the primary disease.13 The patients with the following associated conditions were excluded from the study: (1) clinically apparent steatorrhea in the form of large, bulky, oily stools because any improvement in the nutritional status of patients with steatorrhea would have been attributed to pancreatic enzyme supplementation and not to dietary intervention; (2) cancer of the pancreas; (3) biliary obstruction in the form of deranged liver function test results and dilated bile duct on ultrasound; (4) patients currently undergoing endoscopic or surgical therapy; (5) patients with uncontrolled diabetes; (6) patients with acute exacerbation of pancreatitis; (7) patients with large pseudocyst (>6 cm in size); (8) patients currently consuming alcohol >40 g/day; (9) opioid analgesic addicts; and (10) patients with comorbid conditions like chronic liver disease.

All the patients underwent a battery of tests for the diagnosis of CP and its complications. These tests included the following: hematology and serum biochemistry and imaging, including ultrasound abdomen and CT. If required, MRCP was done.

Nutritional and Dietary Assessment

The patients underwent a detailed nutritional and dietary assessment.

Nutritional assessment was assessed by the anthropometric profile of patients. Anthropometry included BMI, which was calculated by using the formula, Weight (in kg)/Height² (in m). The triceps skinfold thickness (TSF) was measured midway between acromion process of scapula and olecranon process by using skinfold calipers (Harpenden). The mid upper arm circumference (MUAC) was measured in the left upper arm, with a nonstretch tape, at the mid-point between the tip of the shoulder and the tip of the elbow (olecranon process and the acromion). Three readings were recorded for each parameter, and the mean was calculated.

For dietary assessment, a detailed dietary history was obtained from each subject at the time of entry into the study by a trained dietitian through an interview by using a food frequency questionnaire. The frequency of consumption of raw foodstuffs before the onset of disease was elicited. A record of all the food consumed during the past 24 hours was made with the recall method. Nutrient intake was calculated with the 24-hour recall method.14 The result was estimated in accordance with the standard Table of Food Composition in India.15 The amounts of proteins, fats, carbohydrates, and the calories for these were computed.

Protein metabolism was assessed by nitrogen balance and creatinine height index (CHI). Nitrogen balance was calculated by using the formula: [Nitrogen intake/day – Nitrogen output/day]. Nitrogen intake was calculated from 24-hour dietary protein intake (total protein intake/6.25), and nitrogen output was estimated from 24-hour urinary nitrogen excretion by using Kjeldahl’s method and an additional 5 mg/kg of nitrogen for integumental and other losses.16,17

CHI is a ratio of the patient’s 24-hour creatinine excretion and the expected normal creatinine excretion. CHI is calculated with the following formula: [(Measured urinary creatinine × 100)/Ideal urinary creatinine for a given height]. Urinary creatinine is an estimate of body muscle mass calculated as urinary creatinine in grams per 24 hours.

Exocrine and endocrine pancreatic functions were also assessed. Endocrine function was done by using blood sugar measurement (fasting and postprandial). Diabetes was diagnosed on the basis of World Health Organization criteria.18

Exocrine function was assessed by measuring fecal chymotrypsin concentration by spectrophotometric method.19 Stools were collected for 24 hours from patients receiving a normal diet; stools were subsequently homogenized, weighed, and stored at −20°C. Fecal fat was measured to quantify fat loss according to van de Kamer et al.20 Patients were given 1 g/kg fat supplement per day for 3 days before stool collection.

A pain score was devised to assess the severity of abdominal pain in patients with CP. It was calculated on the basis of frequency of pain (No episode of pain in last 12 months = 0/one episode per 3–12 months = 1/one episode per 3 months = 2/one episode per month = 3/two episodes per week or continuous = 6) of CP, and treatment/severity (no treatment = 0/oral analgesics = 2/parenteral analgesics = 4/hospitalization = 6) of the pain.

Randomization

All the study patients were randomly assigned, by using computer-generated random number list, to either of the 2 groups, dietary counseling or dietary supplementation. Random allocation sequence, enrollment, and assigning participants to the 2 groups were done by separate individuals. The participants knew what intervention they were getting, those administering the intervention knew what was being administered, but the person assessing the outcome was blinded to the treatment the patient was receiving.

The daily nutrient requirement of the patients in both groups was calculated on the basis of Harris Benedict equation, which takes into account the present weight, age, sex, and height of the patient, and this value was then multiplied by 1.9 to compensate for hypermetabolic state in chronic disease.21 Patients in both the groups received pancreatic enzyme supplementation (4 capsules 3 times a day to be taken at the start of, during, and at the end of meals). Each enteric-coated microsphere capsule contained lipase 8000 USP, amylase 30,000 USP, and protease 30,000 USP (Digestomen-P; Menarini Raunaq Pharma Limited, India).

In the dietary counseling group, the patient’s usual dietary intake was assessed by 24-hour recall and food frequency questionnaire, and the calorie deficit in the diet was calculated as the
difference between recommended calories and actual intake. An expert dietitian counseled and encouraged the patient to increase the dietary intake by eating small frequent servings of normal homemade diet including all food groups, that is, cereals, pulses, milk, vegetables, fruit, sugar, and oil. No emphasis was placed on using any particular type of oil. A diet chart was prescribed for the required amount of calories. The approximate distribution of calories into the nutrients was carbohydrates 60%, proteins 10%–15%, and fat 25%–30% of total energy intake.

In the dietary supplementation group, the average current daily intake of calories by the patients was calculated, and the deficit in the calorie intake (required minus actual) was supplemented by commercially available polymeric formula feeds (Nutren 1.0; Nestle India Ltd, India). One 250-mL serving of such enteral formula feed provided 250 kcal and 9.5 g proteins along with vitamins, minerals, choline, taurine, and carnitine. The distribution of calories into the nutrients was carbohydrates 51%, proteins 16%, and fat 33% of total energy intake. The feed was MCT-enriched with 25% of fat as MCT. It contained 50% casein and 50% whey as the protein source, had a high level of natural antioxidants, and was lactose-, cholesterol-, and gluten-free. To check for compliance of the patients, they were asked to bring back empty tins of the supplement consumed at every follow-up visit. The calculated dietary intake target was similar in both groups.

**Outcome Measures**

To evaluate improvement in anthropometry, improvement in BMI at 3 months was taken as the primary outcome measure. Secondary outcome measures included TSF, MUAC, dietary intake (the total calorie intake of patient in each group), nitrogen balance, pancreatic exocrine function (assessed by change in fecal fat), and pain score.

The study was approved by the Indian Council of Medical Research. All the patients were included in the study after an informed written consent. The study was conducted in accordance with the humane and ethical principles of research set forth in the Helsinki guidelines. The study followed CONSORT guidelines.

**Sample Size**

In the absence of any previous data on this subject and on the basis of an estimate of around 30–50 new patients of CP

<table>
<thead>
<tr>
<th>Table 1. Clinical and Imaging Features at Baseline: Counseling Versus Supplementation Groups</th>
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<td>Variables</td>
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<tr>
<td>Age (y)</td>
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<tr>
<td>Male sex (n)</td>
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<tr>
<td>Duration of disease (y)</td>
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<tr>
<td>Etiology</td>
</tr>
<tr>
<td>Alcoholic (n)</td>
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<tr>
<td>Idiopathic (n)</td>
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<tr>
<td>Diabetes (n)</td>
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<tr>
<td>Pseudocyst (n)</td>
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<tr>
<td>Pancreatic calcification (%)</td>
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</table>

\(^a\)Mean (standard deviation).

\(P = \text{NS for all variables.}\)
Table 2. Hematology and Biochemical Profile at Baseline in Counseling Versus Supplementation Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Counseling</th>
<th>Supplementation</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>13.21 ± 0.42</td>
<td>13.52 ± 1.23</td>
<td>NS</td>
</tr>
<tr>
<td>Fasting sugar (mg/dL)</td>
<td>112.00 ± 36.00</td>
<td>114.77 ± 72.00</td>
<td>NS</td>
</tr>
<tr>
<td>Serum bilirubin (mg/dL)</td>
<td>0.59 ± 0.10</td>
<td>0.88 ± 0.65</td>
<td>NS</td>
</tr>
<tr>
<td>Serum total protein (g/dL)</td>
<td>7.55 ± 0.64</td>
<td>8.14 ± 0.41</td>
<td>NS</td>
</tr>
<tr>
<td>Serum albumin (g/dL)</td>
<td>4.21 ± 0.44</td>
<td>4.57 ± 0.47</td>
<td>NS</td>
</tr>
<tr>
<td>Serum AST (IU)</td>
<td>43.30 ± 24.93</td>
<td>33.22 ± 18.97</td>
<td>NS</td>
</tr>
<tr>
<td>Serum ALT (IU)</td>
<td>42.95 ± 29.87</td>
<td>29.28 ± 17.21</td>
<td>NS</td>
</tr>
<tr>
<td>Serum alkaline phosphate (IU)</td>
<td>253.21 ± 220.54</td>
<td>140.07 ± 79.29</td>
<td>NS</td>
</tr>
<tr>
<td>Serum calcium (mg/dl)</td>
<td>10.14 ± 0.42</td>
<td>9.50 ± 2.10</td>
<td>NS</td>
</tr>
<tr>
<td>Stool chymotrypsin (unit/g stool)</td>
<td>3.41 ± 2.75</td>
<td>4.42 ± 3.04</td>
<td>NS</td>
</tr>
</tbody>
</table>

per year in our hospital, we took a sample size of 60 patients to be recruited during a 3-year period. A total of 201 patients with CP were seen in our hospital during the 3-year period. Of these, 84 were not malnourished and hence did not meet the inclusion criteria. Of the remaining, 35 patients were excluded because of presence of biliary obstruction (n = 9), clinically apparent steatorrhea (n = 7), comorbidities like chronic liver disease, gastric outlet obstruction, intestinal or pulmonary tuberculosis (n = 4), continued heavy alcohol intake (n = 4), presence of large pseudocyst (n = 3), uncontrolled diabetes mellitus (n = 3), opioid addiction (n = 2), carcinoma of the pancreas (n = 1), acute exacerbation of pancreatitis (n = 1), and patients undergoing endoscopic therapy (n = 1). Twenty-two patients refused to participate. Hence, 60 patients were included in the study (Figure 1, CONSORT chart).

Statistical Analysis

The distributions of fecal fat and fecal chymotrypsin were normalized by log transformation. Intergroup comparison was done with independent sample t test. Intragroup comparison at periods of follow-up was studied by using paired sample t test. All parameters were compared with intention-to-treat analysis. No ancillary analysis was performed. The data are presented as mean (standard deviation) or median (range) as appropriate. A P value of <.05 was taken as significant. All analyses were performed with the SPSS software (SPSS 12.0; SPSS Inc, Chicago, IL).

Results

Baseline Clinical Profile

The clinical characteristics of patients randomized to dietary counseling and to dietary supplementation have been described in Table 1. The mean age of the patients was 30 years (±10); 83% were men. The etiology of CP was alcoholic in 40% and idiopathic in 59%. The baseline hematology and biochemical parameters of patients in both the groups are given in Table 2.

Of the 60 patients studied, 29 were randomized to receive dietary counseling alone, whereas 31 received dietary supplements. There was no evidence of any adverse events in either intervention group. The flow of patients is given in Figure 1.

Effect of intervention on nutritional status. The effect of intervention on nutritional status is shown in Figure 2.

The 2 groups were not different in their anthropometric profile at baseline (Table 3). At the end of 3 months of intervention, there was significant improvement in all the anthropometric parameters including BMI, MUAC, and TSF in both groups. BMI increased from 17.2 ± 1.7 to 18.1 ± 1.8 kg/m² (P = .001) in the dietary counseling group and from 16.7 ± 1.6 to 18.2 ± 1.6 kg/m² (P = .001) in the dietary supplementation group. There was, however, no significant difference between the counseling and supplementation groups at 3 months (18.1 ± 1.8 vs 18.2 ± 1.6 kg/m²; P = NS).

There was no significant difference in the parameters of protein metabolism at baseline in the 2 groups (Table 4). At the end of 3 months, the dietary supplementation group showed significant improvement in protein metabolism as assessed by a positive nitrogen balance and a high CHI. This trend was not seen in those receiving dietary counseling. The 2 groups were, however, still not different in terms of protein metabolism at 3 months.

![Figure 2. Effect of intervention on nutritional status.](image-url)
Table 3. Anthropometric Variables at Baseline and 3 Months: Counseling Versus Supplementation Groups

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Counseling</th>
<th>Supplementation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline (n = 29)</td>
<td>3 Mo (n = 25)</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>47.1 ± 6.3</td>
<td>50.1 ± 7.0</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>17.2 ± 1.7</td>
<td>18.1 ± 1.8</td>
</tr>
<tr>
<td>MUAC (cm)</td>
<td>22.2 ± 2.4</td>
<td>23.4 ± 2.5</td>
</tr>
<tr>
<td>TSF (mm)</td>
<td>7.7 ± 3.6</td>
<td>8.8 ± 5.5</td>
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</table>

NOTE. Values shown as mean ± standard deviation.  
^Paired t test within the group.  
^t test between supplementation and counseling groups at 3 months.

Effect of intervention on dietary intake. There was a significant improvement in the dietary intake of fat, carbohydrates, and proteins as well as total energy in both groups at 3 months (Figure 3, Table 5). In the dietary supplementation group, there were 41.25% and 66.66% increases in the mean calorie and fat intakes, respectively, of which 42.14% of calories and 37.5% of fat were provided by the dietary supplements. There was no difference between the 2 groups in terms of dietary intake at baseline and during subsequent follow up.

Effect of intervention on pancreatic exocrine function. There was a significant decrease in the fecal fat excretion, suggesting an improved fat absorption in both groups at 3 months. Fecal fat decreased from 14.8 to 8.0 g/d (P = .007) in the dietary counseling group and from 12.8 to 6.9 g/d (P = .001) in the dietary supplementation group. There was, however, no significant difference in the fecal fat excretion between the 2 groups at baseline or during subsequent follow up.

Effect of intervention on pain score. The 2 groups showed significant improvement in the pain score during follow-up. It decreased from 3.6 to 3.3 (P = .001) in the dietary counseling group and from 3.0 to 3.4 (P = .001) in the dietary supplementation group. There was, however, no significant difference between the 2 groups at baseline and at the end of 3 months.

Discussion

Patients with CP might experience malnutrition and malnutrition. Chronic inflammation and fibrosis in the gland can destroy exocrine tissue, leading to inadequate delivery of digestive enzymes to the duodenum in the prandial and postprandial periods and subsequent malnutrition. Malnutrition is augmented by inadequate bicarbonate delivery to the duodenum, with secondary inactivation of enzymes and bile acids by gastric acid. Abdominal pain, sitophobia, nausea, vomiting, postprandial satiety, and ongoing alcohol abuse might contribute to poor oral intake. Gastric dysmotility and mechanical gastric outlet obstruction from fibrosis in the pancreatic head might contribute to malnutrition and clinical decline. Patients with CP might at times experience profound steatorrhea and weight loss.

The standard therapy for CP is centered on management of pain. Little emphasis has been placed on adequate nutrition of these patients. Aggressive nutrition therapy in CP might have the ability to change the course of the disease and ameliorate the clinical outcome. Nutrition in CP patients has twin objectives; first and foremost is improvement of the malnutrition, and second is to decrease pain by decreasing pancreatic gland stimulation. Recently, commercially available food supplements have been actively propagated for their easy digestibility and better tolerance for patients with pancreatic insufficiency. The homogenized form of these foods might cause pancreatic stimulation for a shorter period of time as compared with complex solids. They also contain hydrolyzed oligopeptides and MCT, which have been shown to cause minimal pancreatic stimulation by way of decreased secretion of CCK. The efficacy of these food supplements vis-à-vis homemade diet, with regard to their ability to improve the nutritional status and symptomatology of patients with CP, has not been studied.

We found in the present RCT that although both dietary supplementation with commercial food preparations and dietary counseling improved the nutritional status of the patients, commercially available food supplements were no more efficacious than routine dietary counseling for a balanced homemade diet in improving the overall nutritional status of patients. The significant improvement in anthropometric parameters and protein metabolism in the dietary counseling group suggests that dietary counseling might be a more efficacious intervention than routine dietary counseling for improving nutritional status of patients with CP.
pometric parameters, dietary intake, pancreatic exocrine test, and pain scores in both groups was perhaps related to overall improvement in their dietary intake coupled with the standard management for CP in a systematic manner and regular follow-up.

One of the important reasons for malnutrition in CP is the misconstrued belief of patients, sometimes reinforced by physicians, that fat intake should be restricted to a minimum for fear of inducing pain. With simple dietary counseling for a wholesome food intake, we were able to alleviate undernutrition in these patients. Restriction of fat leads to impalatability of food besides decreasing the overall caloric value of the food. Fat intake need not be altered in quantity or modified in quality (use of MCTs) because these patients are receiving adequate pancreatic lipase supplementation by way of exogenous pancreatic enzymes. Moreover, it has been shown that gastric lipase secretion is also increased in CP, and this enzyme can achieve about 30% of the lipolysis observed in healthy volunteers.26,27

The use of MCT in place of long chain triglycerides has been shown to reduce postprandial CCK release.28 CCK, in turn, has been implicated as one of the factors responsible for pancreatic stimulation and subsequent pain in patients with CP.11 However, only a single small study of 10 patients with CP has shown a reduction in pain in patients receiving MCT-enriched food supplements.29 CCK release is dependent on CCK releasing factor (CCK-RF). In the presence of trypsin and other proteases, this releasing factor is easily degraded. However, CP leads to decreased secretion of proteases, leading to loss of the negative feedback on CCK-RF and elevated CCK levels.30 Supplementation with pancreatic enzymes, including proteases, could possibly result in destruction of CCK-RF, so that CCK levels are not high enough to result in pain. Moreover, MCTs also have a low energy density, are not very palatable, and hence have a poor compliance and might induce side effects such as nausea, abdominal pain, and diarrhea.24 Hence, it is usually recommended that MCTs be advised only to those patients who do not gain weight adequately in spite of standard management for CP and those with persistent steatorrhea. It is generally believed that 80% of the patients can be managed by dietary counseling, analgesics, and pancreatic enzyme supplements, whereas only 10%–15% might require oral nutritional supplements.31

The improvement in protein anabolism, as assessed by nitrogen balance, was better in patients on supplements as compared with dietary counseling. However, the difference was not significant at the end of 3 months between the 2 groups, which could be due to a small sample size. The reasons for better nitrogen balance in the intervention group could be related to (1) consumption of adequate proteins through nutritional supplements, (2) low dietary protein intake due to predominantly vegetarian diet in the counseling group, and (3) better assimilation or absorption of oligopeptides present in the supplements.

The weight gain observed in both groups could be attributed to (1) increased calorie, fat, and protein intake; (2) use of exogenous pancreatic enzyme supplements resulting in decreased fecal fat; and (3) decreased pain experienced by patients in both groups, with resultant increase in calorie consumption, attributable to use of pancreatic enzyme supplements and regular patient follow-up. Although the possibility of Hawthorne effect cannot be excluded in our study, it is less likely because there was definite objective improvement in weight gain and nutritional status after the intervention in the groups under study.32 The Hawthorne/protocol effect is a component of the nonspecific benefit from improved routine care within a trial.33 The effect, however, is minimal at best.34

There are several clinical implications of this study. (1) Homemade diet, which is cheaper, more palatable, and more physiologic, improves the nutritional status of patients with CP. (2) Patients with CP should be advised to take small, frequent, homemade balanced meals. Dietary counseling by a trained dietitian should be made an integral part of the management of these patients. (3) Nutritional supplements are not generally required; they might be used only in special situations.

Thus, we concluded that dietary counseling for a balanced homemade diet is as good as commercial food supplements in improving malnutrition in patients with CP.

### Table 5. Dietary Intake at Baseline and 3 Months: Counseling Versus Supplementation Groups

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Counseling</th>
<th>Supplementation</th>
<th>P value&lt;sup&gt;a&lt;/sup&gt;</th>
<th>P value&lt;sup&gt;b&lt;/sup&gt;</th>
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<tr>
<td></td>
<td>Baseline (n = 29)</td>
<td>3 Mo (n = 25)</td>
<td></td>
<td></td>
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<tr>
<td>Energy (kcal/day)</td>
<td>2188 ± 672</td>
<td>2575 ± 141</td>
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<td></td>
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<tr>
<td>Carbohydrates (g/day)</td>
<td>346 ± 140</td>
<td>377 ± 147</td>
<td>.031</td>
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<tr>
<td>Proteins (g/day)</td>
<td>73 ± 24</td>
<td>85 ± 26</td>
<td>.044</td>
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<td>Fats (g/day)</td>
<td>52 ± 24</td>
<td>67 ± 29</td>
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<td>Baseline (n = 31)</td>
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NOTE: Values are shown as mean ± standard deviation.
<sup>a</sup>Paired t test within the group.
<sup>b</sup>t test between supplementation and counseling groups at 3 months.
References


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