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/m^2) 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/m^2, $P = .001$; 16.7 ± 1.6 vs 18.2 ± 1.6 kg/m^2, $P = .001$). 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.[1] 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.[2,3] The etiology of malnutrition in these patients is multifactorial. Maldigestion 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.[4] Self-imposed dietary restriction caused by the fear of inducing pain also contributes to poor nutrition.[5] Physicians also, as a general habit, advise patients with CP to reduce their fat intake to a minimum.[6] 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.[2] 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.[4] Hypermetabolic state with increased resting energy expenditure is another cause of malnutrition in 30%–50% of patients with CP.[7] The degree of undernutrition has a negative impact on the outcome of these patients.[8,9] Micronutrient deficiency might also contribute to decreased antioxidant capacity and increased oxidative stress in these patients.[10]

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.[11] 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.

Abbreviations used in this paper: BMI, body mass index; CCK-RF, CCK releasing factor; CP, chronic pancreatitis; CHI, creatinine height index; CT, computed tomography; ERCP, endoscopic retrograde cholangiopancreatography; MRCP, magnetic resonance cholangiopancreatography; MCT, medium chain triglycerides; MUAC, mid upper arm circumference; RCT, randomized controlled trial; TSF, triceps skinfold thickness.

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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/m^2, 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)/Height^2 (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 foodstuff 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/one episode per week = 4/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