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ORIGINAL ARTICLE

Anthropometric measurements of nutritional status in chronic pancreatitis in India: comparison of tropical and alcoholic pancreatitis

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Abstract
Aim Undernutrition is considered to be a cause of tropical pancreatitis (TP) since this disease is commonly seen in the underprivileged populations of the world. This study was done to compare the nutritional status in patients with TP and alcoholic chronic pancreatitis (ACP) using anthropometric measurements.
Methods Anthropometric measurements were done in patients with TP and ACP aged >18 years and matched healthy controls. Presence of pain, recent dietary restriction, diabetes mellitus (DM), calcification, serum prealbumin (PAB), and quantitative fecal elastase (FE) was assessed. Premorbid body mass index (BMI) was determined from weight before the onset of illness as reported by the patients.
Results Of 54 patients (47 male), 39 (72.2%) had TP and the rest had ACP. Patients with TP were younger than those with ACP; the frequency of pain, DM, calcification, and exocrine insufficiency was similar in the two groups. Compared to control subjects, patients had lower BMI, triceps skin fold thickness (TSFT) and mid-arm circumference (MAC) ($p<0.01$), but waist-to-hip ratio (W/H) was similar. Undernutrition was equally common in TP and ACP (15 [38.5%] vs. 6 [40%]). The BMI, TSFT, MAC, and W/H were similar in TP and ACP. The premorbid BMI was higher than that at presentation (20.2 [3.8] kg/m² vs. 19.1 [3.3] kg/m², $p<0.01$). There was no association between BMI and features contributing to undernutrition (DM, pain, recent dietary restriction, FE level, and calcification) on univariate analysis.
Conclusions Energy undernutrition occurs equally commonly in TP and ACP and this appears to develop after the onset of illness.

Keywords Anthropometry · Fecal elastase · Prealbumin · Tropical calcific pancreatitis · Undernutrition

Introduction

Chronic pancreatitis (CP) is a progressive disease that results in abdominal pain and irreversible exocrine and endocrine deficiency accounting for significant morbidity and healthcare costs [1]. While alcohol is the most common cause of CP in the western world, a non-alcoholic form of CP called tropical pancreatitis (TP) has been described from India and other tropical countries [2, 3]. The initial report from India described this condition as occurring in young malnourished individuals, characterized by severe abdominal pain, pancreatic calcification, and diabetes mellitus (DM) [4]. Because TP has been reported mostly from areas where undernutrition is common, this has long been suspected as a likely cause [5–10]. Undernutrition in TP, however, could be secondary to the dietary restrictions consequent to frequent or persistent abdominal pain, malabsorption of fat and inappropriate dietary advise. The underlying alcoholism, onset of DM or its poor control, and development of adenocarcinoma can also contribute to

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weight loss in CP [11–13]. We hypothesized that energy undernutrition would be equally prevalent in well-matched groups of patients with TP and alcoholic chronic pancreatitis (ACP) if it was the result rather than the cause of the former condition. The present study was undertaken to evaluate the frequency of energy undernutrition in CP using anthropometric measurements and to compare the same between TP and ACP.

Methods

Patients

Patients with CP aged 18 years and above seen at the Department of Gastroenterology and Hepatology, Kasturba Hospital, Manipal from July 2007 to March 2009 were prospectively included. The diagnosis of CP was based on typical clinical features and any one of the following: the presence of pancreatic calcification seen on abdominal radiograph, ultrasonography, computed tomography (CT), or characteristic changes seen on endoscopic retrograde cholangiopancreatography (ERCP), magnetic resonance cholangiopancreatography or endosonography [14]. Patients with hereditary pancreatitis, corrected serum calcium value >11 mg/dL, and those with pancreatic cancer as evidenced by CT scan and/or elevated CA 19–9 levels were excluded from the study, as were those with pseudocysts, ascites, and duodenal or biliary obstruction. The patients were said to have ACP if they drank more than 40 g of alcohol per day for at least five years [15]. TP was diagnosed by excluding other causes of chronic pancreatitis.

Controls

An equal number of age and sex-matched healthy controls were recruited from among the attendants of patients coming to the hospital who had no known health problems or symptoms. They underwent only the anthropometric measurements. The socio-economic status of patients and controls was determined by modified Kuppuswamy's Socioeconomic Status grading [16].

Anthropometric measurements

Height, weight, triceps skin fold thickness (TSFT), mid-arm circumference (MAC), hip circumference (HC), and waist circumference (WC) were measured on patients and controls as adopted from NHANES III nutritional assessment protocol and the body mass index (BMI) and waist-by-hip (W/H) ratio were calculated [17–20]. TSFT was measured to the nearest millimeter with skin calipers and an average of three readings was taken. Energy undernutrition was defined as a BMI of less than 18.5 kg/m² [21]. Patients were interviewed to assess restriction of food intake due to abdominal pain in the recent past and arbitrarily divided into two groups based on the number of days they were on a normal diet over the previous month, that is, ≥15 days and <15 days. In those with onset of disease after the age of 18 years, the body weight before the onset of symptoms was noted from previous health records or as remembered by the patient and the premorbid BMI calculated using the current height.

Blood was collected and the plasma stored at −20°C for measurement of prealbumin (PAB) by a turbidimetric assay (Synchron® Prealbumin, Beckman Coulter India, India). A random stool sample was collected for fecal elastase (FE) assay by Elisa (ScheBo® Pancreatic Elastase 1™ Stool Test, Schebo Biotech, Germany). Exocrine pancreatic insufficiency was classified as absent, mild-to-moderate, or severe based on FE levels of more than 200, 100–200, and less than 100 μg/g stools, respectively [22–24].

The study protocol was approved by the Kasturba Hospital Ethics Committee. Informed consent was obtained from all the patients and controls prior to inclusion into the study.

Statistical analysis

Continuous variables were expressed as mean (SD). SPSS 15.0 was used for statistical analysis. Student's unpaired and paired t tests and the Mann Whitney *U* test were used as appropriate for comparison of continuous data and the Chi square test for comparison of proportions. Pearson's correlation was used to measure the degree of association between two continuous variables.

Results

Subjects

Fifty-four patients (47 (87.0%) men) and an equal number of age and sex-matched controls were included. Thirty-nine (72.2%) patients had TP and 15 (27.8%) patients had ACP. Patients with TP were younger than those with ACP; all patients with ACP were male. The patients and controls, and the subgroups of TP and ACP were comparable in their socio-economic status. The two subgroups with CP were also comparable in all other parameters including FE levels (Table 1).

Fecal elastase (FE)

Forty-two (77.8%) patients with CP had severe exocrine pancreatic insufficiency, eight had (14.8%) mild to moder-

Table 1 Baseline characteristics in patients and controls

		All patients with chronic pancreatitis (n=54)	Tropical pancreatitis (n=39)	Alcoholic chronic pancreatitis (n=15)	Control subjects (n=54)
Age (years) (mean [SD])		34.5 (11.7)	31.0 (10.5)	43.7 (9.6)*	34.5 (11.7)
Male:female		47:7	32:7	15:0	47:7
Socioeconomic status (median [range])		3 (1–4)	3 (1–4)	3 (1–4)	3 (1–4)
Duration of disease (months) (median [range])		13 (1–30)	16 (1–30)	12 (2–24)	–
Abdominal pain		54 (100%)	39 (100%)	15 (100%)	–
Weight loss at presentation		11 (20.4%)	8 (20.5%)	3 (20.0%)	–
Diabetes mellitus		16 (29.6%)	11 (28.2%)	5 (33.3%)	–
Calcification		33 (61.1%)	22 (56.4%)	11 (73.3%)	–
Days on normal diet in the past 30 days (n)	≥15	51 (94.4%)	37 (94.9%)	14 (93.3%)	–
	<15	3 (5.6%)	2 (5.1%)	1 (6.7%)	–
Fecal elastase (μg/gm) (median [range])		–	14.2 (0–316.3)	53.3 (0–145)	–
Prealbumin (mg/dL) (median [range])		–	17 (3.6–35.6)	13.3 (2.6–41.7)	–

*p<0.05 ACP vs. TP

ate insufficiency, and four (7.4%) patients had normal elastase levels.

Anthropometric measurements

The weight, BMI, TSFT, and MAC were lower in patients with CP than in controls (p<0.001, Table 2). Anthropometric measurements were not different between TP and ACP (Table 2). Energy undernutrition was present in 15 (38.5%) patients with TP and 6 (40%) of those with ACP (p=ns). None of the control subjects had undernutrition.

Premorbid weight was available in 40 (74.1%) patients (30 with TP and 10 with ACP). Premorbid BMI was computed using the current height in 35 patients (64.8%) and five patients were excluded as the onset of disease was before the age of 18 years. The premorbid BMI in these 35 patients (21.1 [3.6] kg/m²) was significantly higher than the BMI in the same patients at presentation (19.5 [3.4] kg/m², p<0.001).

Serum prealbumin (PAB)

Thirty-four (62.7%) patients had low serum (PAB) (<18 mg/dL) levels. PAB levels were higher with increasing BMI (r=0.397, p<0.01).

Factors contributing to undernutrition and BMI

The patients with CP were subdivided into two groups based on the presence or absence of each of the factors that might contribute to undernutrition, that is, DM, severe exocrine insufficiency, calcification, more than 15 days off normal diet during the last month, and their BMI at presentation was compared. There was no difference between the two groups for any of the parameters

Table 2 Comparison of anthropometric measurements between patients with chronic pancreatitis (CP) and control subjects

	All patients with CP (n=54)	Tropical pancreatitis (n=39)	Alcoholic chronic pancreatitis (n=15)	Control subjects (n=54)	p-value*
Height (cm)	164.8 (6.5)	164.4 (6.9)	165.9 (5.6)	167.8 (7.5)	>0.05
Weight (kg)	52.4 (10.0)	52.6 (10.6)	52.0 (8.6)	68.5 (4.8)*	<0.001
BMI (kg/m²)	19.2 (3.3)	19.2 (3.3)	19.0 (2.3)	24.6 (1.5)*	<0.001
Triceps skin fold thickness (mm)	9.6 (3.9)	9.9 (4.1)	8.6 (3.9)	12.5 (1.2)*	<0.001
Mid arm circumference (cm)	24.5 (3.3)	24.6 (3.5)	24.3 (3.1)	29.9 (4.3)*	<0.001
Waist:hip ratio	0.91 (0.1)	0.89 (0.1)	0.94 (0.1)	0.92 (0.12)	>0.05

Data represented as mean (SD)

*For TP vs. ACP

mentioned (Table 3). Because of this, further regression analysis was not done.

Discussion

The present study has shown that undernutrition is common in CP and that it appears to develop after the onset of the disease. Being equally prevalent in TP and ACP, it is not unique to the former condition. These findings suggest that energy undernutrition may not be the cause of TP, but its effect. Our results support and extend those of a previous study on patients with idiopathic CP from subtropical India [15].

Nutritional factors have always been considered to be important in the etiology of CP [25–27]. Energy undernutrition has long been, and often still is considered to be the cause of TP [28]. The discovery of mutations in several genes in various types of CP and other recent advances in our understanding of the etiology of this condition appear to have undermined the importance of nutritional factors as cause; in recent years, these have been implicated as possible modifiers of other important causative factors [29].

PAB has a half-life of two days and hence reflects more recent changes in the nutritional status than do anthropometric measurements [30]. In our patients, undernutrition was common as assessed by serum PAB levels and BMI, and these parameters correlated well. This suggests a poor energy intake in the immediate past as well as over a medium term. Interestingly, the W/H ratio was not different between patients and control subjects. This could reflect premorbid accumulation of fat within the abdomen which, unlike in other parts of the body, is not easy to loose even in nutritionally compromising situations. All these observations again suggest that energy undernutrition in TP may be the result rather than the cause of the disease.

Abdominal pain that limits food intake in the earlier stages of the disease, and pancreatic exocrine and endocrine deficiency in later stages, are important contributors to weight loss in CP. All patients in the present study had abdominal pain at presentation. The TP and ACP groups were well-matched in terms of factors that might have contributed to undernutrition, such as socioeconomic status, abdominal pain, calcification, DM, and exocrine insufficiency. We excluded patients with complications, such as pancreatic cancer, pseudocysts, ascites, and duodenal obstruction specifically to avoid the variable nutritional effects of these complications.

Table 3 Body mass index according to the presence of factors contributing to undernutrition

Parameters		*N*	BMI (Kg/m²)* (mean [SD])
Diabetes mellitus	Present	16	19.16 (3.51)
	Absent	38	19.14 (3.24)
Fecal elastase (μg/g)	<100	42	19.19 (3.26)
	100–200	8	20.14 (3.70)
Calcification	Present	33	19.45 (3.50)
	Absent	21	18.65 (2.95)
Days off normal diet in past one month (n)	≥15	51	19.20 (3.37)
	<15	3	18.30 (1.44)

p = ns for all

We found no association between energy undernutrition and the factors that are thought to contribute to it. The reasons for this could be many. The small numbers making up the subgroups analyzed is one obvious reason. DM and increased proportion of calories from proteins in diet have been associated with weight loss in patients with idiopathic CP [15]. Resting energy expenditure is raised in CP [31]. There might be other as yet unexplored factors that could contribute to weight loss in patients with CP, and these might differ depending on the stage of the disease [32, 33]. More studies are needed to understand these aspects better. A larger sample size would have helped us define the factors that contributed to undernutrition in these patients. However, it should be noted that defining these factors was not the primary aim of our study.

Whatever the factors contributing to it might be, energy undernutrition is common in CP. The nutritional status is an important factor determining the outcome in sick, hospitalized patients [34–37]. CP being a condition that entails considerable morbidity and often needing surgical or non-surgical interventions, this nutritional aspect needs much more attention. Unfortunately, the nutritional status in CP has received far less attention than that in acute pancreatitis [13, 38, 39].

While our results suggest that energy undernutrition may be the effect rather than the cause of TP, this study was not designed to prove or disprove such an etiological association. A prospective cohort study would prove such an association or its lack. We did not calculate a sample size and hence our results might reflect a type 2 error. However, only 38.5% of our patients with TP were undernourished. The premorbid BMI was calculated from the premorbid weight as reported by the patients. Such data have inherent biases. Also, weight loss following the onset of disease as shown by us does not preclude pre-existing undernutrition. These facts should be taken into account in interpreting our results.

The pattern of TP has been changing in the recent years. Patients present at an older age now, are better nourished, and live longer sometimes to present with cancer as a complication [40]. While on an average these patients are younger at presentation than those with ACP,

the gap between them is narrowing [15, 41, 42]. Clearly TP cannot be confined to the narrow description given by Geeverghese in the 1960s of young malnourished patients presenting with "abdominal pain in childhood, diabetes in adolescence, and death in the prime of life". In fact, TP has also been reported from subtropical regions in the Indian subcontinent [43, 44]. It is interesting to speculate that TP and the idiopathic CP described from the West might represent a disease continuum and that the differences between these conditions reflect differences in the availability of health care facilities, nutritional and antioxidant status, or the genetic make-up of the respective populations [45, 46]. Further studies are needed to answer these questions and better understand this enigmatic disease.

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