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Original article

The nutritional status and factors contributing to malnutrition in children with chronic pancreatitis

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A B S T R A C T

Background/objectives: The present study was undertaken to determine the prevalence of malnutrition among children with chronic pancreatitis (CP). Furthermore, we aimed to evaluate the relationship between etiological factors of CP, its clinical characteristics, and the severity of malnutrition.
Methods: The study included 208 children with CP (113 girls and 95 boys; mean age: 10.8 years, range: 1.6–18 years), hospitalized at our center between 1988 and 2012. The severity of malnutrition was graded on the basis of Cole's ratios, and its prevalence was analyzed according to the etiological factors of pancreatitis. Moreover, the analysis of discrimination was performed to identify the factors contributing to malnutrition among the following variables: age at CP onset, duration of CP, number of CP exacerbations, the number of ERCPs performed, the grade of pancreatic damage documented on imaging, co-occurrence of diabetes, and the results of 72-h fecal fat quantification.
Results: We documented features of malnutrition in 52 (25%) children with CP, including 36 (17.3%) patients with moderate malnutrition, and 2 (0.96%) with severe malnutrition. There was no significant difference in the prevalence of malnutrition between groups of patients with various etiological factors of chronic pancreatitis. The age at CP onset showed the best discrimination ability of malnourished patients: the mean age at disease onset in a subgroup of malnourished children was significantly higher than in children with Cole's index >85%.
Conclusions: A considerable percentage of children with CP can suffer from clinically significant malnutrition. Later age at CP onset predisposes to development of malnutrition.

1. Introduction

Chronic pancreatitis (CP) is a disease that rarely occurs in childhood. It is characterized by continuous damage of pancreatic structure, function, or both, caused by progressive inflammation. Patients with CP often suffer from progressive multifactorial malnutrition caused by decreased dietary intake, malabsorption, surgical procedures and recurrent infections. The recommendations regarding nutrition in children are still empirical due to lack of prospective trials in pediatric patients with chronic pancreatitis. Pediatric studies investigating energy expenditure, nutritional status, bone metabolism, and body composition in children with chronic pancreatitis are not available.

Children with CP rarely develop symptoms of endocrine and exocrine pancreas insufficiency. However, it should be remembered that the pancreas is an organ that does not regenerate and any further aggravation impairs its function, to a lesser or greater extent. Symptoms of previously latent pancreatitis typically escalate with age. Between 50% and 80% of adult patients with chronic pancreatitis develop pancreatic exocrine insufficiency. Pancreas has a large functional reserve, and usually only after the destruction of more than 90% of the glandular tissue, does a distinct impairment of digestion of fats, proteins and carbohydrates occur. This leads to malabsorption syndrome, initially manifested by steatorrhea and azotorrhea [1–4]. Furthermore, diabetes can develop at later stages of CP, due to the loss of insulin producing beta-cells in the pancreas [5].

There are only few studies assessing the nutritional status of patients with chronic pancreatitis. All published research concerns adult patients, mainly with chronic alcoholic pancreatitis, whereas dietary recommendations for children with CP are extrapolated

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Table 1
Diagnostic criteria for chronic pancreatitis [9].

	Diagnostic procedure	Definite chronic pancreatitis	Probable chronic pancreatitis
1a.	US	Pancreatic stones evidenced by intrapancreatic hyperreflective echoes with acoustic shadows behind	Intrapancreatic coarse hyperreflectivities, irregular dilatation of pancreatic ducts, or pancreatic deformity with irregular contour
1b.	CT	Pancreatic stones evidenced by intrapancreatic calcifications	Pancreatic deformity with irregular contour
2.	ERCP	1) Irregular dilatation of pancreatic duct branches of variable intensity with scattered distribution throughout the entire pancreas, or 2) Irregular dilatation of the main pancreatic duct and branches proximal to complete or incomplete obstruction of the main pancreatic duct (with pancreatic stones or protein plugs)	Irregular dilatation of the main pancreatic duct alone; intraductal filling defects suggestive of non-calcified pancreatic stones or protein plugs
3a.	Secretin test	Abnormally low bicarbonate concentration combined with either low enzyme outputs or low secretory volume	Abnormally low bicarbonate concentration alone, or low enzyme output plus low secretory volume
3b.	Tubeless tests:	–	Simultaneous abnormalities in the BT-paraaminobenzoic acid test and fecal chymotrypsin test, observed at two points in time several months apart
4.	Histologic examination:	Irregular fibrosis with destruction and loss of exocrine parenchyma in tissue specimens obtained at biopsy, surgery or autopsy. The fibrosis has an irregular and patchy distribution in the interlobular spaces. Intralobular fibrosis alone is not specific for chronic pancreatitis	Intralobular fibrosis with one of the following findings: loss of exocrine parenchyma, isolated islets of Langerhans, or pseudocysts.
5.	Others	Additionally, protein plugs, pancreatic stones, dilatation of the pancreatic duct, hyperplasia and metaplasia of ductal epithelium and cyst formation are observed.	

The following criteria apply primarily to patients with the symptoms and findings suggestive of chronic pancreatitis. A diagnosis of chronic pancreatitis is made when one or more of these criteria are met. In asymptomatic patients confirming examinations should be repeated several months later. The diagnostic modalities listed are arranged in the sequence in which they are used in routine practice and are independent of each other.

from studies in adults [5–8]. As a leading national pancreatic center, The Children's Memorial Health Institute in Warsaw admits most of the pediatric chronic pancreatitis cases from Poland. Our group of children with chronic pancreatitis (over 200 patients) is one of the largest single-medical-center series in the world. This gave us the possibility of objective assessment of nutritional status in children with CP.

The present study was undertaken to determine the prevalence of malnutrition on the basis of anthropometric measurements of children with chronic pancreatitis. Furthermore, we aimed to evaluate the relationship between etiological factors of chronic pancreatitis, its clinical characteristics, and the severity of malnutrition.

2. Methods

A total of 208 children with CP (113 girls and 95 boys), hospitalized at the Department of Gastroenterology, The Children's Memorial Health Institute in Warsaw, between 1988 and 2012, were enrolled into the study. Mean age of the participants was 10.8 years (range: 1.6–18 years). The etiological factors of CP and the course of the disease were established retrospectively based on the clinical data (medical and family history, nutritional status, laboratory parameters, genetic testing, imaging studies: MRCP and ERCP, surgical and endoscopic treatment, pancreatic enzyme therapy). Chronic pancreatitis was diagnosed on the basis of diagnostic criteria proposed by Homma et al. [9] (Table 1). The first episode of acute pancreatitis, diagnosed on the basis of serum amylase activity ≥3 times over the upper normal range (reference value: 0–82 U/L), elevated urine amylase activity (reference value: 0–380 U/L), and serum lipase activity ≥5 times over the upper normal range (0–210 U/L), was regarded as the onset of CP.

The following inclusion criteria for participation in the study were set: 1) age ≤18 years, 2) features of CP verified by one of the imaging methods (ERCP, MRCP, CT or US scan) and/or by tests evaluating exocrine pancreatic insufficiency (the 72-h fecal fat quantification, elastase-1 stool test, breath test with ^{13}C-mixed substrates estimating exocrine pancreatic function), and 3) observation period of ≥1 year from the first episode of pancreatitis. The exclusion criteria were: 1) age >18 years, 2) lack of imaging studies or the absence of CP features in imaging studies and functional tests, 3) cystic fibrosis 4) inability of long-term observation of CP process (single visit in outpatient clinic or single hospitalization).

The nutritional status was analyzed according to the etiological factors of pancreatitis. All patients were divided into 5 groups, depending on the etiological factor: 1) patients with hereditary pancreatitis (*PRSS1* gene mutation and/or family history allowing the diagnosis of hereditary pancreatitis, $n = 26$), 2) patients with gene mutations predisposing to pancreatitis (*CFTR* and/or *SPINK1*), in whom other potential cause of the disease was not found ($n = 46$), 3) patients with anatomic anomalies of the pancreatic duct, confirmed during hospitalization, in whom other potential cause of the disease was not identified ($n = 20$), 4) patients with two or more coexisting etiologic factors of CP (excluding *PRSS1* gene mutation, $n = 24$), and 5) patients with idiopathic CP ($n = 92$). Patients with lipid disorders, biliary tract diseases, autoimmune pancreatitis, history of abdominal injury or infection, or possible post-drug etiology were incorporated into the latter group due to lack of a certain causative relationship with the development of pancreatitis. Mean follow-up for all the groups was 5 years.

Body Mass Index (BMI) was calculated from the formula: $BMI = actual\ weight\ [kg]/(height\ [m])$ [2]. The calculated value of BMI was compared with the data of the centile chart. The rate of BMI, calculated using growth charts (BMI percentile), allows an adequate assessment of the nutritional status of patients <18 years

Table 2
Values of Cole's ratio with corresponding nutritional status.

Cole's ratio (%)	Nutritional status
<75	Severe malnutrition
75–85	Malnutrition
86–90	Mild malnutrition
91–110	Normal
111–120	Overweight
>120	Obesity

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