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Original article

The role of high fat diet in the development of complications of chronic pancreatitis☆

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SUMMARY

Background: Little is known about risk factors for complications in chronic pancreatitis (CP). High fat diet (HFD) has been demonstrated to aggravate pancreatic injury in animal models. The aim of this study was to investigate the role of HFD in age at diagnosis of CP and probability of CP related complications.
Methods: A cross-sectional case–case study was performed within a prospectively collected cohort of patients with CP. Diagnosis and morphological severity of CP was established by endoscopic ultrasound. Pancreatic exocrine insufficiency (PEI) was diagnosed by ^{13}C mixed triglyceride breath test. Fat intake was assessed by a specific nutritional questionnaire. Odds ratios (OR) for CP related complications were estimated by multivariate logistic regression analysis.
Results: 168 patients were included (128 (76.2%) men, mean age 44 years (SD 13.5)). Etiology of CP was alcohol abuse in 89 patients (53.0%), other causes in 30 (17.9%) and idiopathic in the remaining 49 subjects (29.2%). 24 patients (14.3%) had a HFD. 68 patients (40.5%) had continuous abdominal pain, 39 (23.2%) PEI and 43 (25.7%) morphologically severe CP. HFD was associated with an increased probability for continuous abdominal pain (OR = 2.84 (95%CI, 1.06–7.61)), and a younger age at diagnosis (37.0 ± 13.9 versus 45.8 ± 13.0 years, $p = 0.03$) but not with CP related complications after adjusting for sex, years of follow-up, alcohol and tobacco consumption, etiology and body mass index.
Conclusions: Compared with a normal fat diet, HFD is associated with a younger age at diagnosis of CP and continuous abdominal pain, but not with severity and complications of the disease.
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1. Introduction

Chronic pancreatitis (CP) is characterized by chronic inflammation of the pancreas ultimately leading to fibrosis and parenchymal destruction. Chronic abdominal pain, exocrine insufficiency leading to maldigestion and malnutrition, diabetes, pseudocysts, bile duct and duodenal strictures and vascular thrombosis are complications associated with the disease,[1] which have a profound and negative influence on quality of life.[2] During the last decades, advances have been made in the understanding of risk factors for CP. Several genes associated with the disease have been described and the association between smoking and pancreatitis has been acknowledged.[3–5] Established risk factors now include alcohol, smoking, genetic mutations, autoimmune pancreatitis, recurrent acute pancreatitis and mechanical obstruction of the pancreatic duct.[6] However, only a fraction of subjects with high alcohol consumption will develop CP[7,8] and the etiology still remains unknown in some cases of CP. Little is known about factors associated with

Abbreviations: PEI, pancreatic exocrine insufficiency; CP, chronic pancreatitis; ^{13}C-MTG, ^{13}C-mixed triglycerides; s-MRCP, secretin enhanced magnetic resonance cholangiopancreatography.

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complications in CP. Smoking has been demonstrated to be associated with pancreatic calcifications,[3,9,10] pancreatic exocrine insufficiency,[3] diabetes[9] and pain in CP.[13] However, studies on other risk factors are scarce.

Dietary fat is the strongest nutritional stimulant of pancreatic secretion.[12] Long-term high fat diet has been demonstrated to induce pancreatic injury in rat models[13,14] and to potentiate alcohol-mediated pancreatic injury.[15] However, few studies have investigated the association between high fat diet and the risk of CP or CP-related complications.[16] The aim of the present study was to investigate the association between high fat diet and the risk of CP related complications at the time of diagnosis of the disease and to compare the age at diagnosis between subjects reporting a high fat diet with those reporting a normal fat diet.

2. Patients and methods

2.1. Design

A cross-sectional case–case study was performed within a prospectively collected cohort of patients with CP at the Pancreas Unit of the Department of Gastroenterology, University Hospital of Santiago de Compostela, Spain. The Ethical Committee for Clinical Research of Galicia approved this study and all participants gave their written informed consent.

2.2. Patients

Patients diagnosed with CP at our gastroenterology out-patient clinic have been prospectively included at the time of diagnosis in a specific database since 2007. For the present study, patients with age <18 years at index visit to the CP outpatient clinic were included. Patients who clearly modified the diet before the index visit were excluded. A modification of the diet was defined as the elimination of any type of food because of symptoms that the patient perceived as related to the disease. The diagnosis of CP was established by endoscopic ultrasonography (EUS). EUS findings were classified according to the Wiersema classification, evaluating 10 criteria for CP (five parenchymal and five ductal).[17] The CP diagnosis was considered as confirmed in the presence of five or more criteria.[18] Patients with 3–4 criteria were further examined by secretin enhanced magnetic resonance cholangiopancreatography (s-MRCP) and pancreatic parenchymal magnetic resonance imaging (MRI) with intravenous injection of gadolinium in order to confirm or exclude the diagnosis. In these patients, the presence of any of the following findings on s-MRCP and MRI was considered to support the EUS finding: Dilation of at least three side branches, dilation of the main pancreatic duct, irregular contour of the main pancreatic duct, delayed and reduced contrast enhancement of the gland or reduced secretin-stimulated pancreatic secretion.[19] Etiology of CP was assessed according to the TIGAR-O classification (toxic, idiopatic, genetic, autoimmune, recurrent acute pancreatitis and obstructive).[6]

2.3. General clinical data

Demographic and clinical data were collected in a detailed questionnaire including sex, age, age from onset of symptoms, age at first episode of acute pancreatitis if any, body mass index (BMI), clinical presentations of the disease (pain, chronic diarrhea or jaundice), smoking and drinking habits, and results of diagnostic imaging. Patients were classified as never smokers (smoked <100 cigarettes in lifetime) or ever smokers (smoked >100 cigarettes). Ever smokers were categorized as past (no use of tobacco during the past 36 months) or current smokers.[5] Average daily alcohol consumption in grams was assessed in the questionnaire. For the purposes of the study, CP was considered as alcohol-related if patients reported a mean daily alcohol consumption >60 g/day.[1]

2.4. Assessment of chronic pancreatitis related symptoms and complications

A detailed abdominal pain history was taken including type (continuous or intermittent), location (epigastrium or hypochondrium) and intensity. Continuous abdominal pain, regardless of severity, has been demonstrated to be the type of pain that is associated with the most important impairment of quality of life and highest rate of disability, hospitalization and medication use in CP.[21] Consequently, continuous abdominal pain, regardless of location or intensity, was used as the outcome when pain as a symptom was analyzed in the present study. Chronic diarrhea was defined according to ROME III classification and classified as present or not.[22] At inclusion, exocrine pancreatic function was investigated in all patients using the ^{13}C-mixed trygliceryde (^{13}C-MTG) breath test.[20] The ratio of $^{13}CO_2/^{12}CO_2$ in exhaled air was measured by mass spectrometry. Pancreatic exocrine insufficiency (PEI) was defined as a 6 h cumulative $^{13}CO_2$-recovery rate of less than 29%.[20] PEI was categorized as present or not. Diabetes mellitus diagnosis was established based on clinical history and serum glucose levels according to the American Diabetes Association guidelines.[23] Morphological severity was made by endoscopic ultrasonography (EUS) according to the Wiersema classification.[16] The morphological picture was classified as severe in the presence of calcifications and/or 7 or more EUS criteria. In cases without calcifications, EUS morphological severity was classified as moderate or mild in the presence of 5–6 and 3–4 criteria, respectively. The presence of duodenal strictures, pseudocysts, bile strictures and mesenteric or portal thrombosis was evaluated during the index EUS examination using endoscopic image, standard B-mode EUS and color Doppler EUS.

2.5. Dietary assessment

Dietary data were obtained through a specific nutritional questionnaire presented to patients at diagnosis.[24] The questionnaire assessed foods consumed during the preceding month. Daily fat and caloric intake was calculated based on standard nutritional contents of different foods and meals according to the Spanish Food Composition Database (Supplement 1). Fat intake was classified as high when the daily caloric intake in fat exceeded 30% of the total daily intake of calories recommended by US Food and Nutrition Board (Table 1).[25] In addition, we specifically asked if patients modified their diet from onset of CP symptoms.

2.6. Statistical methods

Clinical characteristics are presented as frequency and percentage of categorical variables and mean and SD of numerical variables. Association between age at onset of symptoms and age at diagnosis and high fat diet was analyzed using Student's *t*-test. Analyses were repeated stratifying for etiology (alcohol related CP, idiopathic CP and other CP etiologies) and in subjects with less than 2 years between onset of symptoms and study entry. Multiple linear regression was used to calculate the association between age at diagnosis and high fat diet adjusting for sex, alcohol and tobacco consumption and time between symptom onset and assessment of CP related complications. Sex, smoking habits, alcohol consumption, diabetes, etiology of CP, morphological severity, PEI, continuous abdominal pain and diarrhea were compared between non-high fat diet and high fat diet cases using chi-square test.

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