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Hyper-Homocysteinemia: A Potential Indicator of Acute Pancreatitis

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Abstract

Objectives: Hyper-homocysteinemia, reportedly, is closely associated with some known vascular dysfunctions, thrombosis and even inflammations. However, till now rare studies were directed to the association between hyper-homocysteinemia and acute pancreatitis (AP). The illumination of this potential association is expected to benefit therapies of AP. In this regard, this study aims at digging the underlying association between hyper-homocysteinemia and AP.

Methods: In this study, 160 patients with AP were involved. The plasma levels of homocysteine of these patients were measured. The patients were divided into two groups, based on the serum homocysteine concentration. Patients with high plasma homocysteine concentrations (C>15 μmol/L) were classified as Group 1, while the rest with normal plasma homocysteine concentrations (5 μmol/L ≤ C ≤ 15 μmol/L) were identified as Group 2. Then, the lipid levels for the two groups were studied to detect whether there existed a synergistic effect of hyper-homocysteinemia and hyper-triglyceride on AP.

Results: Almost half (46.9%) of the patients with AP were found to bear hyper-homocysteinemia in this study. However, patients in Group 1 were found to have lower triglyceride and total cholesterol levels than those in Group 2, with P=0.038 and P=0.046 respectively. In addition, more patients in Group 2 had hyper-triglyceride and hyper-total cholesterol than those in Group 1, though the differences were not significantly observed.

Conclusions: In conclusion, these studies showed that hyper-homocysteinemia may be a very important indicator of a high risk of AP. So, more attention should be paid to hyper-homocysteinemia in the progress of AP.

Keywords: Homocysteine; AP; Chinese population

Introduction

AP (AP) is a common disease that features auto-digestion of the pancreas, and is the second highest cause of total hospital stays, the largest contributor to aggregate costs, and the fifth leading cause of in-hospital deaths [1-4]. Furthermore, patients with pancreatitis reportedly bear higher risk of developing pancreatic cancer [5,6]. Biliary disease and excessive alcohol consumption are the most common reasons for AP, but acute and chronic recurrent pancreatitis have also been reported in patients with varieties of concurrent metabolic disorders involving hyperlipidaemias, branched-chain amino acid degradation, homocystinuria, haemolytic, acute intermittent porphyria and several amino acid transporter defects [7]. For instance, hyperlipidaemia is one of the most common causes of AP, and triglyceride (TG) levels above 11.3 mmol/L (1000 mg/dl) are generally considered to put patients at high risks of developing pancreatitis [8,9]. Elevated plasma homocysteine concentrations had been reported to involve the development of arterial thrombosis and atherosclerosis [10]. Based on this point, high homocysteine levels may also be one cause of AP, endothelial dysfunction and impaired microvascular circulation. However, to date, little research was focused on the relationship between plasma homocysteine concentrations and AP [11,12]. Hence, we began our studies on the relationship between plasma homocysteine concentrations and AP and possibilities of the synergistic effect of plasma homocysteine levels and triglyceride levels on the occurrence of AP.

Materials and Methods

Subjects

Patients who met at least two of the three following clinical standards were diagnosed as AP bearers: abdominal pain; serum amylase above the upper reference limit of more than 3 times; positive ultrasonography or computed tomography scan. 160 patients (103 men, 57 women) diagnosed with AP in Henan Provincial People's Hospital, Henan Province, China, were recruited for this study. The study was approved by the ethical committees of Henan Provincial People's Hospital. Informed consents were obtained from all patients.

Methods

Information such as the tobacco habits and alcohol consumption of each individual was obtained from the records in the hospital or by face-to-face interviews. Blood samples of all the patients were taken within 24 hours after admission to the hospital. Serum levels of triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and homocysteine were measured on an automatic biochemical analyzer (AU5400, Olympus Optical Co, Sizuoka, Japan). The patients were divided into two groups (Group 1 and Group 2) based their serum homocysteine concentration levels. Patients with high plasma homocysteine concentrations (C>15 μmol/L) were classified as Group 1 (50 men, 25 women), while the rest with normal plasma homocysteine concentrations (5 μmol/L ≤ C ≤ 15 μmol/L) were identified as Group 2 (53 men, 32 women). The clinical characteristics of Group 1 and 2 were shown in Table 1. Differences

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	Group 1 (n=75)	Group 2 (n=85)	P
Age (year)	45.1 ± 15.7	41.5 ± 14.9	0.14
BMI (kg/m^2)	25.4 ± 4.0	25.8 ± 4.6	0.66
Smoking (n, %)	27 (36.0%)	37 (43.5%)	0.42
Alcohol drinker (n, %)	26 (34.7%)	34 (40.0%)	0.52
Homocysteine (μmol/L)	26.1 ± 13.9	10.7 ± 2.8	---
TG (mmol/L)	3.0 ± 4.0	4.6 ± 5.4	0.038
TC (mmol/L)	4.7 ± 1.8	5.5 ± 2.9	0.046
HDL-C (mmol/L)	1.1 ± 0.5	1.3 ± 1.0	0.15
LDL-C (mmol/L)	2.8 ± 1.3	2.9 ± 1.6	0.51
Patients with hyper-TG (n, %)	39 (52.0%)	56 (65.9%)	0.079
Patients with hyper-TC (n, %)	19 (25.3%)	30 (35.3%)	0.23
Diabetes (n, %)	1 (1.3%)	6 (7.1%)	0.12
Hypertension (n, %)	4 (5.3%)	13 (15.3%)	0.069

Table 1: Clinical characteristics of the patients in different groups.

between groups were analyzed using Student's *t* test for continuous data and chi-square test for categorical data. The differences were regarded significant if the *P* value did not exceed 0.05. All analysis was performed using SPSS 19.0.

Results

The association between hyper-homocysteinemia and AP

The results were shown in Table 1. Among all the 160 patients with AP, 75 patients (46.9%, Group 1) which made up a very high percentage were identified with increased plasma homocysteine levels. There were no significant differences of age, BMI, smoking or/and drinking status, serum HDL-C and LDL-C levels between the two groups.

The association between hyper-homocyteinemia and hyper-TG

Notably, the patients in Group 1 (bearing higher plasma homocysteine levels) had lower serum TG and TC levels compared with patients in Group 2 (bearing normal plasma homocysteine levels), with P=0.038 and P=0.046 respectively. In the contrast, there were more patients in Group 2 bearing hyper-TG and hyper-TC than in Group 1 (65.9%vs 52.0%, and 35.3%vs 25.3% respectively), despite that the differences were not large(P=0.079 and P=0.23 respectively).

Discussion

Among the 160 patients with AP, 59.4% of patients (95 persons) got hyper-TG, which was consistent with the previous research that people with hyper-TG were at high risk of getting AP [8,9,13]. And 46.9% of patients (75 persons) had higher plasma homocysteine levels, which, very possibly, indicated that higher plasma homocysteine levels could also increase the risks of suffering AP. This kind of association was rarely deeply studied and reported. Previously, Girish reported that chronic pancreatitis was associated, to some extent, with hyper-homocysteinemia, and that low folate levels maybe a key role in this derangement [12]. Yuzbasioglu reported that patients with AP had higher plasma homocysteine levels than subjects without AP [11]. The discovery in our studies that 46.9% of involved patients got hyper-homocysteinemia, was really consistent with the previous reports, and further demonstrated that a high level of plasma homocysteine is a risk factor for AP. And this high level of plasma homocysteine should be paid enough attention in the prevention of AP.

Moreover, we found that hyper-homocysteinemia and hyper-TG had no observed synergistic effects in patients with AP. Patients with hyper-homocysteinemia generally had lower TG and TC levels than those with normal homocysteine levels. According to some early reports [12,14], 5, 10-methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism and low folate levels were supposed to be the main mechanism behind the up regulation of hyper-homocysteinemia in AP. Based on the mechanism, proper folate supplementation is helpful to lower homocysteine levels, resulting in the prevention of AP. And further confirmation of this hypothesis needs more studies to be done in the future.

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