The relationship between triglyceride level and the severity of acute hypertriglyceridemic pancreatitis in Chinese patients

Ya Mei Sun, Feng Gao*, Xue Chen, Jie Zhang*

Department of Digestive, Beijing Anzhen Hospital, Capital Medical University, Beijing, China

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ABSTRACT

Background/Aims: *The aim of this study was to investigate the relationship between the triglyceride (TG) level and the severity of acute hypertriglyceridemic pancreatitis (AHTGP) in Chinese patients.*

Materials and Methods: *On the basis of clinical data on AHTGP, patients from the period 2015-2018 were enrolled retrospectively and grouped according to the 2012 revised Atlanta classification. Kruskal-Wallis test was performed to evaluate differences among groups. Receiver operating characteristic (ROC) curves were generated to assess the ability of parameters to distinguish mild acute pancreatitis (MAP)/moderately severe acute pancreatitis (MSAP) from severe acute pancreatitis (SAP).*

Results: *A total of 104 patients with AHTGP were enrolled and divided into three groups: 61 patients with MAP, 29 patients with MSAP, and 14 patients with SAP. The median values for the MAP, MSAP, and SAP groups were as follows: TG level 48 h after admission, 3.4, 4.5, and 14.2 mmol/L, respectively ($p<0.001$); ratio of TG level 48 h after admission to that 0 h after admission (48:0-h ratio), 19.4, 32.1, and 65.9, respectively ($p<0.001$). ROC curves showed that the areas under the curves for the TG level 48 h after admission and the TG 48:0-h ratio for predicting SAP were 0.965 and 0.917, respectively ($p<0.001$), and the optimal cut-off values were 7.8 mmol/L and 37.7, respectively.*

Conclusion: *The TG level 48 h after admission and the TG 48:0-h ratio may predict the severity of AHTGP, and a high TG level 48 h after admission may be correlated with the progression of SAP.*

Keywords: *Acute hypertriglyceridemic pancreatitis, triglyceride, severity*

Introduction

Acute hypertriglyceridemic pancreatitis (AHTGP) is the most common form of recurrent acute pancreatitis (RAP). A markedly elevated triglyceride (TG) level (≥11.3 mmol/L) is a well-established etiology of acute pancreatitis (AP), being present in 2%-4% of patients with AP (1). In China, the incidence of AHTGP is increasing more rapidly than that of alcoholic AP, and AHTGP is now the second most common etiology of AP; indeed, the incidence of AHTGP among patients with AP in a multicenter study in Beijing was 10.36% (2). However, most scoring systems evaluating the severity of AP do not include the TG level (3-6), and the results of clinical studies assessing the relationship between the TG level and the severity of AHTGP are conflicting (1,6-9). Therefore, we investigated the relationship between the TG level and the severity of AHTGP in Chinese patients.

Materials and Methods

We retrospectively analyzed clinical data from patients diagnosed with AP and hospitalized at Beijing Anzhen Hospital, Capital Medical University (Beijing, China), between 2015 and 2018. Patients with missing baseline clinical data at the onset of AP and the interval time more than 24 h between the onset of abdominal pain and admission were excluded. The TG 48:0-h ratio was calculated as the ratio of the TG level at 48 h after admission to that 0 h after admission, multiplied by 100. All patients were instructed to fast after admission.

This study received ethical approval from the Local Ethics Board of Beijing Anzhen Hospital, Capital Medical University (number 2015022X).

Definitions

The diagnostic criteria for AHTGP were a serum TG level ≥11.3 mmol/L and two or more of the following three symptoms: acute upper abdominal pain with backward radiating pain, amylase or lipase level higher than threefold the upper limit of normal, and abdominal computed tomography findings indicative of AP (3,10). The exclusion criteria were other etiologies of AP and RAP.

**Ya Mei Sun and Feng Gao contributed equally to this work.*

Corresponding Author: ***Jie Zhang; zhangjie4155@sina.com***

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The 2012 revised Atlanta classification was adopted to evaluate the severity of hypertriglyceridemic pancreatitis (HTGP) (3). The absence of organ failure and local or systemic complications was considered to indicate mild acute pancreatitis (MAP). The presence of transient (within 48 h) organ failure and/or local complications or exacerbation of comorbid disease was regarded as moderately severe acute pancreatitis (MSAP). The presence of persistent (>48 h) organ failure was considered to indicate severe acute pancreatitis (SAP). The respiratory, cardiovascular, and renal systems were assessed to identify organ failure, which was defined as a modified Marshall score >2 for one of these three systems. Peripancreatic fluid collection, pancreatic and peripancreatic necrosis (sterile or infected), pseudocyst, and walled-off necrosis (sterile or infected) were defined as local complications. Exacerbation of a preexisting comorbidity, such as coronary artery disease or chronic lung disease, precipitated by AP was defined as a systemic complication.

The AP scoring systems (3,11,12) included the Ranson score, based on 11 items (age, serum glucose level, white blood cell [WBC] count, aspartic transaminase, and lactate dehydrogenase after admission, serum levels of hematocrit, calcium, partial pressure of arterial oxygen [PaO_2], base deficit, blood urea nitrogen [BUN], and fluid sequestration 48 h after admission); the bedside index for severity in acute pancreatitis (BISAP), comprising five items (BUN level, impaired mental status, systemic inflammatory response syndrome, age, and pleural effusion); the Acute Physiology and Chronic Health Evaluation II (APACHE II), involving an acute physiology evaluation, age, and chronic health evaluation; and the modified computed tomography severity index (MCTSI), which comprises pancreatic inflammation, pancreatic necrosis, and extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, and gastrointestinal tract involvement).

MAIN POINTS

- *Current results of clinical studies assessing the relationship between the serum triglyceride (TG) level and the severity of acute hypertriglyceridemic pancreatitis (AHTGP) are conflicting.*
- *This study showed patients with AHTGP of differing severity had similar TG levels 0 h after admission. However, the TG level 48 h after admission and the TG 48:0-h ratio was significantly positively correlated with the severity of AHTGP, and the predictive power was comparable to that of the MCTSI score, APACHE II, Ranson and BISAP scores.*
- *Therefore, The TG level 48 h after admission and the TG 48:0-h ratio may predict the severity of AHTGP.*

The BISAP and APACHE II scores were calculated 24 h after admission, and the Ranson score was calculated 48 h after admission. The MCTSI score was calculated for patients who had undergone contrast-enhanced computed tomography (CT) within 48 h of admission.

Comparative Groups

AHTGP patients were divided into MAP, MSAP, and SAP groups. AHTGP patients were also categorized into lower and higher groups based on the TG level 0 h after admission, TG level 48 h after admission, and the TG 48:0-h ratio; boundary values of 22.6 mmol/L, 5.6 mmol/L, and 30.0, respectively, were used (1,6,13-15).

Statistical Analysis

Categorical data were coded as numbers and compared using the chi-squared test or Fisher's exact test. Non-normally distributed continuous data are expressed as medians (interquartile ranges) and were compared by the Kruskal-Wallis test. A stepwise linear regression analysis was performed to assess the influences of the independent variables on the severity of AHTGP while controlling for the effects of other variables. In the linear regression analysis, the dependent variable was the severity of AHTGP and the independent variables were age, gender, and the results of clinical laboratory tests (alanine aminotransferase, aspartate aminotransferase, albumin, total bilirubin, direct bilirubin, alkaline phosphatase, glutamyltranspetidase, lactate dehydrogenase, BUN, serum creatinine, glucose, total cholesterol, calcium, and amylase) at 0 h after admission. Spearman's correlation analysis was used to assess correlations. The ability of the TG level to distinguish MAP/MSAP from SAP was assessed using receiver operating characteristic (ROC) curves followed by calculation of the areas under the curve (AUCs) and 95% confidence intervals. These results were used to determine the optimal cut-off values for TGs based on a tradeoff between sensitivity and specificity. A $p<0.05$ was considered significant. All data were analyzed using the Statistical Packages for the Social Sciences (SPSS) software ver. 21.0; (IBM Corp., Armonk, NY, USA).

Results

Demographic and Clinical Characteristics of Patients with AHTGP

A total of 104 patients with AHTGP (61 with MAP, 29 with MSAP, and 14 with SAP) were enrolled. The demographic and clinical characteristics of these patients are shown in Table 1. The three groups did not differ in

Table 1. Demographic and clinical characteristics of the patients with acute hypertriglyceridemic pancreatitis (AHTGP).

Item	MAP (n=61)	MSAP (n=29)	SAP (n=14)	p (Kruskal-Wallis, chi-squared or Fisher's exact test)
Age, median (IQR), years	41.0 (35.0-48.5)	41.0 (32.5-47.0)	36.0 (26.0-45.0)	0.434
Male/Female (n)	46/15	20/9	11/3	0.809
BMI, median (IQR), kg/m2	27.7 (26.0-29.7)	28.0 (25.7-29.8)	29.7 (27.5-36.7)	0.115
Hospital time, median (IQR), day	8.0 (6.0-9.0)	11.0 (8.5-14.0)	15.5 (10.5-19.0)	< 0.001
Diabetes mellitus, n (%)	11 (18.0)	5 (17.2)	3 (21.4)	0.639
Hypertension, n (%)	13 (21.3)	6 (20.7)	3 (21.4)	1.000
Coronary heart disease, n (%)	2 (3.3)	2 (6.9)	1 (7.1)	0.518
Fatty liver, n (%)	55 (90.2)	26 (89.7)	13 (92.9)	1.000
TG level 0 h after admission, median (IQR), mmol/L	17.5 (12.8-25.1)	14.0 (12.0-25.6)	23.4 (17.1-37.4)	0.062
TG level 48 h after admission, median (IQR), mmol/L	3.4 (2.5-6.1)	4.5 (3.3-6.2)	14.2 (11.5-21.1)	<0.001
TG 48:0-h ratio, median (IQR)	19.4 (14.6-28.5)	32.1 (17.0-47.0)	65.9 (43.0-70.8)	<0.001
TC level 0 h after admission, median (IQR), mmol/L	6.8 (6.0-8.8)	6.0 (5.5-9.0)	11.4 (8.1-15.6)	<0.001
TC level 48 h after admission, median (IQR), mmol/L	5.4 (4.4-6.4)	4.6 (4.0-6.8)	6.5 (6.0-8.3)	0.023
TC 48:0-h ratio, median (IQR)	79.3 (65.3-87.2)	78.1 (67.5-83.3)	67.7 (51.3-79.9)	0.105
WBC 0 h after admission, median (IQR), 109/L	10.4 (7.4-13.3)	14.0 (11.3-18.9)	17.1 (14.1-18.9)	<0.001
CRP 0 h after admission, median (IQR), mg/L	10.8 (4.0-44.7)	46.8 (16.2-110.5)	149.5 (46.0-222.7)	<0.001

TG: triglyceride; MAP: mild acute pancreatitis; MSAP: moderately severe acute pancreatitis; SAP: severe acute pancreatitis; BMI: body mass index; TC: total cholesterol; WBC: white blood cell; CRP: C-reactive protein; IQR: interquartile range.

Table 2. Results of stepwise linear regression analysis (r^2 = 0.617).

Item	Unstandardized coefficients	p
Constant	0.253	0.070
TG level 48 h after admission (mmol/L)	0.037	<0.001
WBC 0 h after admission (109/L)	0.053	<0.001
CRP 0 h after admission (mg/L)	0.003	<0.001
TG 48:0-h ratio	0.007	0.008

TG: triglyceride; WBC: white blood cell; CRP: C-reactive protein.

terms of age; gender; body mass index; or medical history of diabetes mellitus, hypertension, coronary heart disease, or fatty liver. The findings for the MAP, MSAP, and SAP groups were as follows: TG level 0 h after admission, 17.5 (12.8-25.1), 14.0 (12.0-25.6), and 23.4 (17.1-37.4) mmol/L, respectively (p=0.062); TG level 48 h after admission, 3.4 (2.5-6.1), 4.5 (3.3-6.2), and 14.2 (11.5-21.1) mmol/L, respectively (p<0.001); and TG 48:0-h ratio, 19.4 (14.6-28.5), 32.1 (17.0-47.0), and 65.9 (43.0-70.8), respectively (p<0.001).

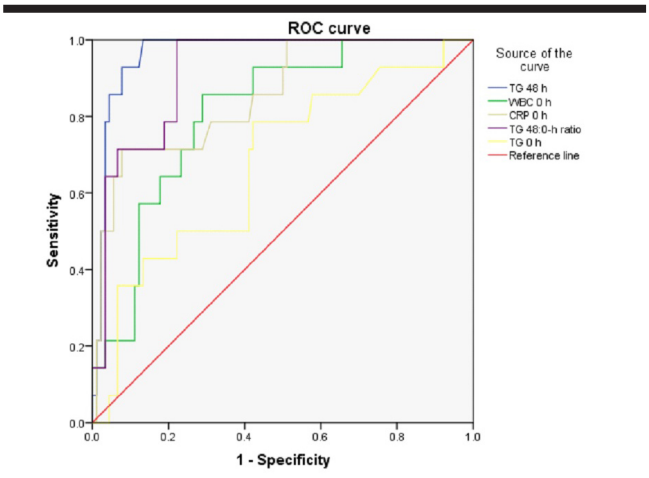
Results of Stepwise Linear Regression Analyses

The results of stepwise linear regression analyses of the associations of demographic and clinical parameters with the severity of AHTGP are shown in Table 2. The TG level at 48 h, WBC count, C-reactive protein (CRP) level, and TG 48:0-h ratio was positively associated with the severity of AHTGP. The linear regression equation was severity of AHTGP=0.253+0.037×TG 48 h+0.053×WBC 0 h+0.003×CRP+0.007×TG 48:0-h ratio. The inde-

Table 3. Spearman's correlation analysis between the triglyceride level and scores on acute pancreatitis scoring systems.

Items		TG level 0 h after admission	TG level 48 h after admission	TG 48:0-h ratio
Atlanta classification	Correlation coefficient	0.062	0.451	0.486
	p	0.532	< 0.001	< 0.001
Ranson score	Correlation coefficient	0.274	0.427	0.235
	p	0.119	0.479	0.449
BISAP score	Correlation coefficient	0.119	0.479	0.449
	p	0.230	< 0.001	< 0.001
APACHE II score	Correlation coefficient	0.025	0.426	0.449
	p	0.804	< 0.001	< 0.001
MCTSI	Correlation coefficient	−0.009	0.464	0.533
	p	0.927	< 0.001	< 0.001

TG: triglyceride; BISAP: bedside index for severity in acute pancreatitis; APACHE II: acute physiology and chronic health evaluation II; MCTSI: modified computed tomography severity index.



Items	AUC	SE	95% CI	*P*-value	Cut-off value
TG 48 h after admission	0.965	0.017	0.931–0.998	$P < 0.001$	7.8
WBC 0 h after admission	0.810	0.054	0.704–0.915	$P < 0.001$	13.9
CRP 0 h after admission	0.854	0.054	0.748-0.960	$P < 0.001$	124.5
TG 48:0-h ratio	0.917	0.030	0.858-0.977	$P < 0.001$	37.7
TG 0 h after admission	0.675	0.078	0.523–0.827	$P = 0.035$	18.0

Figure 1. Receiver operating characteristic (ROC) curves and the corresponding areas under the curve (AUCs) for the triglyceride (TG) level, white blood cell (WBC) count, and C-reactive protein (CRP) level for prediction of severe acute pancreatitis (SAP) among patients with acute hypertriglyceridemic pancreatitis (AHTGP).

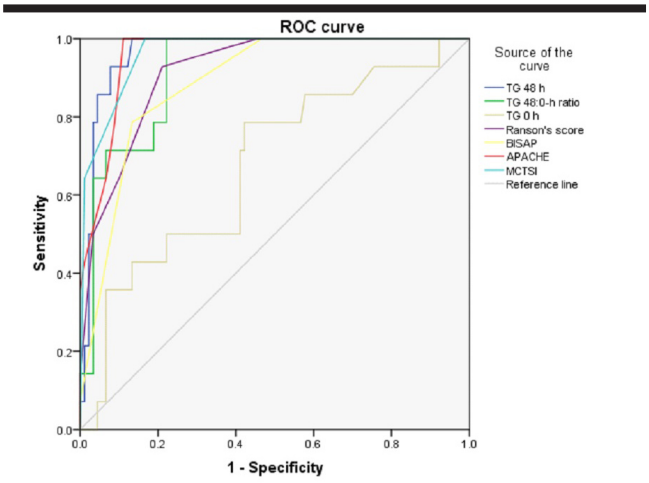
pendent variables age (p=0.398), gender (p=0.485), alanine aminotransferase (p=0.967), aspartate aminotransferase (p=0.711), albumin (p=0.399), total bilirubin (p=0.691), direct bilirubin (p=0.814), alkaline phosphatase (p=0.088), glutamyltranspetidase (p=0.594), lactate dehydrogenase (p=0.158), BUN (p=0.778), serum creatinine (p=0.083), glucose (p=0.244), TG level 0 h after admission (p=0.263), total cholesterol (p=0.471), pH (p=0.309), PaO_2 (p=0.059), calcium (p=0.562), and amylase (p=0.798) were not associated with the severity of AHTGP.

Ability of the TG Level at 48 h to Predict the Severity of AHTGP

The Spearman's correlation analysis results are shown in Table 3. The TG level 48 h after admission and the TG 48:0-h ratio was significantly positively correlated with the Atlanta classification, Ranson, BISAP, APACHE II, and MCTSI scores.

The AUCs from the ROC analyses of TG level for predicting SAP in AHTGP are shown in Figure 1. The AUCs of the TG level 48 h after admission, WBC 0 h after admission, CRP 0 h after admission, TG 48:0-h ratio, and TG level 0 h after admission were 0.965, 0.810, 0.854, 0.917, and 0.675, respectively, and the optimal cut-off values were 7.8, 13.9, 124.5, 37.7, and 18.0, respectively. The AUCs of the TG level 48 h after admission, MCTSI score, APACHE II score, TG 48:0-h ratio, Ranson score, BISAP score, and TG level 0 h after admission were 0.965, 0.965, 0.959, 0.917, 0.916, 0.888, and 0.675, respectively (Figure 2). The TG level 48 h after admission had the largest AUC, indicating that its predictive power was comparable to that of the MCTSI score and superior to that of the APACHE II, Ranson, and BISAP scores.

The relationship between the TG level and the severity of AHTGP is shown in Figure 3. TG levels were classified as higher or lower than predetermined boundary values. The severity of AHTGP did not differ significantly be-



Items	AUC	SE	95% CI	*P*-value
TG 48 h after admission	0.965	0.017	0.931–0.998	$P < 0.001$
TG 48:0-h ratio	0.917	0.030	0.858-0.977	$P < 0.001$
TG 0 h after admission	0.675	0.078	0.523–0.827	$P = 0.035$
Ranson's score	0.916	0.032	0.854-0.978	$P < 0.001$
BISAP	0.888	0.038	0.814-0.962	$P < 0.001$
APACHE	0.959	0.018	0.923-0.994	$P < 0.001$
MCTSI	0.965	0.017	0.931-0.999	$P < 0.001$

Figure 2. Receiver operating characteristic (ROC) curves and corresponding areas under the curve (AUCs) for the triglyceride (TG) level, Ranson score, bedside index for severity in acute pancreatitis (BISAP), acute physiology and chronic health evaluation II (APACHE), and modified computed tomography severity index (MCTSI) for prediction of severe acute pancreatitis (SAP) among patients with acute hypertriglyceridemic pancreatitis (AHTGP).

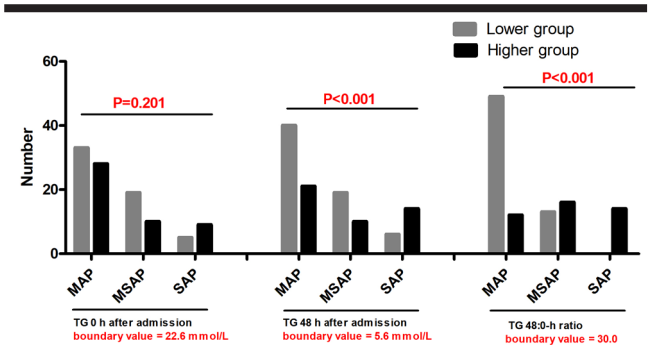


Figure 3. Relationship between the triglyceride (TG) level and the severity of acute hypertriglyceridemic pancreatitis (AHTGP).

tween the groups with higher and lower TG levels 0 h after admission (boundary value=22.6 mmol/L). However, the groups with a higher TG level 48 h after admission (boundary value=5.6 mmol/L) and a higher TG 48:0-h ratio (boundary value=30.0) contained greater proportions of patients with SAP than did the groups with lower values for these parameters.

Discussion

Hypertriglyceridemia (HTG) is the most common etiology of recurrent RAP. The clinical characteristics of HTGP are similar to those of AP of other etiologies. AHTGP is typically seen in patients with one or more secondary factors (uncontrolled diabetes, alcohol abuse, special medications/oral estrogen, and certain pregnancy durations) and abnormal lipoprotein metabolism (familial hyperlipidemia or HTG). The frequency of HTGP as the etiology of AP is increasing worldwide (3,16). The mechanism by which HTG causes HTGP is unknown; however, the most widely accepted hypothesis is that hydrolysis of excess TG by pancreatic lipase results in the accumulation of free fatty acids (FFAs). FFAs can injure acinar cells and pancreatic capillaries, resulting in ischemia. Ischemia, in turn, induces an acidic environment and further enhances the toxicity of FFAs. Another theory is that a high concentration of chylomicrons results in hyperviscosity in pancreatic capillaries, which can lead to ischemia and acidosis in the pancreas. Furthermore, the pathogenesis of HTGP also involves endoplasmic-reticulum stress (6,17-19). However, the risk of AP in the presence of serum TG levels >11.3 mmol/L and > 22.6 mmol/L is ~5% and ~10%-20%, respectively (1). In addition, the results of clinical studies assessing the relationship between the TG level and the severity of AHTGP are conflicting (1,6-9), and it is unclear whether patients with TG levels >22.6 mmol/L suffer more severe AP.

In our study, patients with AHTGP of differing severity had similar TG levels 0 h after admission, and the TG level 0 h after admission was weakly correlated with the scores of AP scoring systems, except for the Ranson score (TG level 0 h after admission was significantly correlated with the glucose, lactate dehydrogenase, and calcium levels). In addition, we found no difference in AP severity between the higher TG group (TG level 0 h after admission >22.6 mmol/L) and the lower TG group. These results suggest that the TG level 0 h after admission is not predictive of the severity of AHTGP. However, patients with SAP had significantly higher TG levels 48 h after admission and TG 48:0-h ratios, and the groups with a higher TG level 48 h after admission (>5.6 mmol/L) and a higher TG 48:0-h ratio (>30.0) had significantly higher incidences of SAP and stronger correlations with the various AP scores. In addition, the cut-off values for the TG level 48 h after admission and the TG 48:0-h ratio for the prediction of SAP were 7.8 mmol/L and 37.7, respectively. Therefore, a high TG level at 48 h after admission without a rapid decline upon fasting was correlated with the progression of SAP.

In our study, the TG level 0 h after admission was not correlated with the severity of AHTGP (p=0.062). However, the TG level 48 h after admission was correlated with the severity of AHTGP (p<0.001). The serum TG level is a reflection of dietary intake, endogenous production, and clearance of TG containing lipids and lipoproteins. Thus, the TG level 0 h after admission is affected by diet composition and other factors, which fluctuate greatly, and the TG level reached a steady state after fasting for 48 h. An ROC analysis was performed to assess the ability of the serum TG level to distinguish SAP from MAP and MSAP, in which the AUC for TG level 48 h after admission (0.965) was greater than that for TG level 0 h after admission (0.675). Therefore, after 48 h fasting, the TG level is predictive of the severity of AHTGP.

The serum TG level usually declines rapidly after fasting owing to diminished influx of TG-rich chylomicrons into the bloodstream, and is further decreased by the administration of hypocaloric intravenous fluids, which cut off the very low-density lipoprotein output from the liver (1). Therefore, a high TG level 48 h after admission and high TG 48:0-h ratio is indicative of increased FFA accumulation in the pancreas and hyperviscosity in the pancreatic capillaries, which can lead to the injury of pancreatic acinar cells and severe ischemia of pancreatic capillaries. In addition, a high TG level 48 h after admission and high TG 48:0-h ratio suggests the need for hypolipidemic treatment (*e.g.*, oral lipidlowering agents, insulin infusion, heparin infusion, and therapeutic plasma exchange). We typically prescribe fibrates (Fenofibrate) and insulin infusion for these patients to increase the activity of lipoprotein lipase, which eliminates TG by catalyzing its degradation into glycerol and FFA.

Most AP-severity scoring systems do not include the TG level (3,11,12), but instead evaluate the glucose level, WBC count, aspartic transaminase activity, lactate dehydrogenase activity, BUN, PaO_2, calcium level, and creatine level. In this study, Spearman's correlation analysis showed that the TG level 48 h after admission and the TG 48:0-h ratio was significantly positively correlated with the Ranson, BISAP, APACHE II, and MCTSI scores. ROC analyses also indicated that the ability of the TG level 48 h after admission to predict the severity of AHTGP was comparable to that of the MCTSI score and superior to that of the APACHE, Ranson, and BISAP scores.

A variety of clinical and laboratory parameters can be used to predict the severity of AP (20-22). We performed a stepwise linear regression analysis of the associations of several independent variables with the severity of AHTGP. The results showed that the TG level at 48 h, WBC count, CRP level, and TG 48:0-h ratio were positively associated with the severity of AHTGP. These results were confirmed by the AUC values of these variables.

Three studies (13-15) have stressed the utility of therapeutic plasma exchange for the treatment of severe HTG. Therapeutic plasma exchange effectively and quickly removes TG from very low-density lipoproteins and chylomicrons from serum and reduces the generation of FFAs, which exerts local and systemic effects; it has been suggested that this treatment should be performed as soon as possible, at 24-48 h intervals, until the TG level decreases to less than 5.6 mmol/L. These studies supported our results that persistent high TG level 48 h after admission without a rapid decline on fasting is usually correlated with progression of SAP.

The treatment of AHTGP AP includes pancreatic rest by bowel rest, fasting, intravenous hydration, and analgesics (23). Other etiologies of AP should be differentiated by performing physical examinations, by conducting periodic laboratory testing, by abdominal imaging and chest CT, by assessing medications (thiazide diuretics, azathioprine, glucocorticoids, and other agents), and by taking and reviewing past patient histories. As previously mentioned, if the TG level does not rapidly decline on fasting 48 h after admission, and there is evidence of SAP (persistent [i.e., >48 h] organ failure) and a need for prolonged fasting, pancreatic rest via postligament of Treitz three-cavity nasojejunal nutrition is considered in our department (usually 5-7 days after admission). We also monitor blood sugar (by finger-stick) and guide insulin infusion. However, we do not have any experience with heparin infusion or therapeutic plasma exchange in the treatment of severe HTG.

Our study had some limitations. All subjects were recruited from one center, which may have resulted in selection bias, the small number of patients might have limited the statistical power of the study, and might have failed to provide TG level 48 h after the onset of abdominal pain. However, this study is, to our knowledge, the first to show that the TG level 48 h after admission and the TG 48:0-h ratio may be predictive of the severity of AHTGP in Chinese patients.

In conclusion, the TG level 48 h after admission and the TG 48:0-h ratio may be predictive of the severity of AHTGP. A high TG level 48 h after admission may be

correlated with the progression of SAP, but future multicenter studies including large samples are needed to further verify this relationship.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Beijing Anzhen Hospital, Capital Medical University, approval no. 2015022X on August 13, 2015.

Informed Consent: Written informed consent was obtained from the patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – Y.M.S., F.G., J.Z.; Design - Y.M.S., F.G., J.Z.; Supervision – J.Z.; Resource - Y.M.S., F.G., X.C.; Materials - Y.M.S., F.G., X.C.; Data Collection and/or Processing - Y.M.S., F.G., X.C.; Analysis and/or Interpretation - Y.M.S, F.G.; Literature Search - Y.M.S., F.G.; Writing - Y.M.S., F.G.; Critical Reviews - J.Z.

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Conflict of Interest: The authors have no conflicts of interest to declare.

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