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Early prediction of persistent organ failure by serum apolipoprotein A-I and high-density lipoprotein cholesterol in patients with acute pancreatitis

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ABSTRACT

Background: Early identification of acute pancreatitis (AP) patients at high-risk of developing persistent organ failure (persistent OF) is a vital clinical goal. This research intends to assess the ability of apolipoprotein A-I (APO A-I) and high-density lipoprotein cholesterol (HDL-C) to predict persistent OF.

Methods: Between January 2011 and September 2016, a total of 102 adult AP patients with organ failure, local complications or deterioration of former comorbidities disease during hospitalization were included in this study retrospectively. Serum lipids were tested and computed the correlation with clinical outcomes or scoring systems. The AUCs to predict persistent OF were also calculated and compared with each other.

Results: Serum APO A-I and HDL-C levels were negatively associated with scoring systems. Meanwhile, serum lipids were negatively correlated with poor clinical outcomes. The AUCs of APO A-I, HDL-C, the combination of APO A-I and BISAP, or the combination of APO A-I and MCTSI to predict persistent OF among Moderately severe acute pancreatitis (MSAP) and Severe acute pancreatitis (SAP) patients were 0.886, 0.811, 0.912, and 0.900 or among those with organ failure were 0.915, 0.859, 0.933, and 0.933, respectively.

Conclusions: The concentrations of APO A-I, HDL-C, and the combinations of APO A-I and scoring systems have high predictive value to predict persistent OF.

1. Introduction

Acute pancreatitis is a common gastrointestinal emergency. Approximately 20% of AP patients ultimately develop to persistent OF. It has a high risk of death ranging from 15% to 25% [1]. A lot of investigations have pointed out that persistent OF was also associated with poor clinical outcomes [1–3]. Conversely, the prognosis is much better in patients with transient OF and disease-related death is seldom reported. Therefore, some investigations suggested to differentiate persistent OF from transient OF and redefine the severity of AP based on the occurrence of persistent OF or not [4]. According to the Revised Atlanta Classification [4,5], AP can be divided into mild acute pancreatitis (MAP), MSAP, and SAP.

It has been well recognized that patients with critical illness would benefit from early management on admission [6]. Moreover, it's tactful to divide AP patients into different groups and adjusting therapy according to the severity of the disease. Making an accurate assessment for the severity of AP at admission is a challenge yet. This is why a great number of AP-related researches are carried out for early assessment of its severity [7]. It was reported critical illness can cause the change of serum lipids in the early stage [8,9]. Moreover, the previous studies showed that lipoprotein was associated with the severity of AP [10–12]. It was noted the levels of APO A-I and HDL-C dropped when the patients developed to severe sepsis. However, the correlation between the severity of AP and APO A-I or HDL-C obtained during the first 48 h of hospitalization has not been extensively discussed using the Revised Atlanta Classification. Otherwise, none of these researches have reported the ability of the combination of APO A-I and scoring systems to predict the occurrence of SAP.

The purpose of this study was to assess the ability of APO A-I, HDL-C, or the combinations of APO A-I and scoring systems to predict persistent OF in AP patients. We further evaluated the association of serum lipids with the poor clinical outcomes in this study. Some scoring systems used for evaluation of the severity of AP were taken in our analysis

Abbreviations: SIRS, systemic inflammatory response syndrome; BISAP, bedside index for severity in AP; MCTSI, modified CT severity index; MMS, modified Marshall score

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Table 1
Comparison of clinical characteristics, laboratory parameters, outcomes and scoring systems between subgroups in patients with AP.

	POF/SAP (n = 26)	MSAP (n = 76)	TOF (n = 40)	P1 SAPvsMSAP	P2 POFvsTOF
Median age, years	39(35–52)	54(38–62)	55(46–62.5)	0.039	0.004
Male sex	18(69)	43(57)	22(55)	0.256	0.248
HTN	6(23.1)	27(36)	16(40)	0.241	0.154
Diabetes on admission	7(26.9)	13(17)	6(15)	0.276	0.234
Hepatic steatosis	15(57.7)	35(46)	21(52.5)	0.305	0.679
Smoking habit	9(34.6)	22(29)	14(35)	0.588	0.974
Median hospital days	19(9.5–27)	15(11–18)	14.5(10–17)	0.173	0.083
Median prehospital days	1(1–2)	1(1–2)	1(0.85–1)	0.193	0.074
Etiology, Alcohol/Biliary/HTG/Others	3/5/12/6	4/18/20/34	3/10/7/20	0.11	0.048
TC, mmol/L	6.47(3.43–9.11)	4.66(3.91–8.33)	4.48(3.77–6.59)	0.833	0.379
TG, mmol/L	8.07(1.21–12.14)	1.33(0.73–8.57)	1.11(0.67–4.50)	0.02	0.006
HDL-C, mmol/L	0.60(0.50–0.79)	0.99(0.72–1.23)	1.05(0.81–1.30)	< 0.001	< 0.001
LDL-C, mmol/L	1.67(1.10–2.09)	2.08(1.52–2.62)	2.08(1.67–2.62)	0.012	0.012
APO A-I, g/L	0.75(0.68–0.83)	1.02(0.91–1.26)	1.03(0.92–1.22)	< 0.001	< 0.001
APO B, g/L	0.78(0.61–1.04)	0.79(0.65–0.91)	0.75(0.62–0.90)	0.997	0.748
Lipoprotein (a), mg/dL	4.20(1.93–10.15)	5.35(2.60–15.83)	5.75(2.30–13.65)	0.562	0.694
APO A/APO B	0.96(0.72–1.39)	1.30(1.02–1.84)	1.33(1.05–1.76)	0.003	0.003
Scr, μmol/L	97.0(55.5–257.5)	61.0(49.0–73.5)	62.0(51.2–72.0)	0.006	0.017
Ca, mmol/L	1.85(1.56–2.07)	2.18(2.03–2.30)	2.19(2.06–2.30)	< 0.001	< 0.001
Bun, mmol/L	8.6(4.3–13.8)	4.8(3.7–5.9)	5.0(3.6–5.9)	0.001	0.004
Pleural effusion	22(84.6)	50(65.8)	21(52.5)	0.069	0.007
Necrosis	8(30.8)	5(6.6)	1(2.5)	0.001	0.001
Infected necrosis	2(7.7)	0(0)	0(0)	0.015	0.075
Pseudocyst	2(7.7)	5(6.6)	1(2.5)	0.846	0.322
Mortality	5(19.2)	0(0)	0(0)	< 0.001	0.004
Impaired mental status	7(26.9)	1(1.3)	0(0)	< 0.001	0.001
Number of organ failures,1/2/3	17/6/3	39/1/0	39/1/0	< 0.001	0.002
SIRS	21(80.8)	33(43.4)	19(47.5)	0.001	0.007
BISAP	3(2–3)	1(1–2)	1(1–2)	< 0.001	< 0.001
MCTSI	5.5(4–8)	4(2–4)	4(2–4)	< 0.001	< 0.001
MMS	4(3.8–5)	2(0–2)	2(2–3)	< 0.001	< 0.001

Categorical variables are described as N (%). Continuous variables are described as median and interquartile range (IQR). POF, persistent organ failure; TOF, transient organ failure; MSAP, Moderately severe acute pancreatitis; SAP; Severe acute pancreatitis; HTG, hypertriglyceridemia; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; APO A-I, apolipoprotein A-I; APO B, apolipoprotein B; Scr, serum creatinine; Ca, calcium; Bun, blood urea nitrogen; SIRS, systemic inflammatory response syndrome; BISAP, bedside index for severity in AP; MCTSI, modified CT severity index; MMS, modified Marshall score.

and the results were compared with APO A-I as well as HDL-C in the prediction of SAP.

2. Materials and methods

2.1. Patient population

We reviewed all medical records of patients diagnosed as AP from January 2011 to September 2016 in our hospital. The present study retrospectively analyzed adult AP patients with organ failure, deterioration of former comorbidities disease or local complications during hospitalization. And they were admitted to in-patient department within a median of 24 h (IQR 24 to 48 h) after the onset of symptoms. All these patients with available serum lipids during the first 48 h of hospitalization during this period were admitted in this research. Patients of chronic pancreatitis and patients taking lipid-lowering medicine were excluded. This study was performed according to the Helsinki Declaration and approved by the Ethics Committee of our hospital.

2.2. Definition

AP diagnosis was made by two or three of the following characteristics: typical clinical features, the concentrations of serum amylase or lipase reached at least three-fold of the upper normal limit, and the typical findings of images [4]. AP patients were diagnosed as MSAP if they had at least one of the following features: transient OF (< 48 h), deterioration of former comorbidities disease or local complications including acute peri-pancreatic fluid collections, pseudocysts, acute necrotic collections, and walled-off necrosis. And patients were diagnosed as SAP if they experienced persistent OF (≧48 h).

According to modified Marshall score [13], organ failure is classified as the score not < 2 of these organ systems (respiratory, pulmonary, and cardiovascular) during hospitalization. SIRS [14] was classified as not less than two following features: temperature 36 < °C or > 38 °C, heart rates > 90 beats/min, white blood cell count < $4 * 109$/L or > $12 * 109$/L, and $PaCO_2$ < 32 mm Hg or respiratory rates > 20/min.

2.3. Data collection and laboratory test

Demographics, comorbidities, and etiology were recorded for each patient on admission. The sample of serum lipids were obtained during the first 48 h of hospitalization and analyzed immediately. The levels of serum calcium were measured using the worst values during hospitalization. The concentrations of these laboratory parameters were all measured using an auto-analyzer (H-7600; Hitachi, Tokyo, Japan). The poor clinical outcomes were evaluated during hospitalization. Additionally, for modified Marshall score (MMS), the most abnormal value at the same time was recorded. Bedside index for severity in AP (BISAP) were calculated using the worst values acquired at the first 24 h of hospitalization. Contrast-enhanced CT were carried out within 48 h after admission and subsequently modified CT severity index (MCTSI) were calculated. All images were read by junior radiologists and reviewed by senior radiologists.

2.4. Statistical analysis

By using the Kolmogorov-Smirnov test, the normal distributions of all continuous variables were tested. Then they were summarized using