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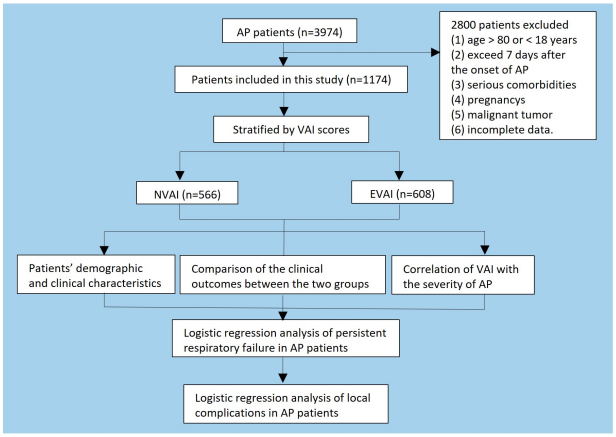
Visceral Adiposity Index as a Key Predictor of Severity in Acute Pancreatitis: A Large-Scale Retrospective Cohort Study

Study population & Methods



Outcomes

- Elevated visceral adiposity index (EVAI) was strongly associated with the severity and local complications of acute pancreatitis (AP).
- EVAI emerged as the most influential independent risk factor for persistent respiratory failure, acute peripancreatic fluid collection and acute necrotic collection.
- Visceral adiposity index (VAI) can be easily calculated through anthropometric measures and laboratory indexes.

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Visceral adiposity index as a key predictor of severity in acute pancreatitis - A large-scale retrospective cohort study

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Authors' contributions

TTW, FC and JJH collected the data, performed statistical analysis, drafted the manuscript, and contributed equally to this work. LZ, XXY, SLM, QPZ, YHL, CTY and CWC participated in the data collection. JL checked the data and edited the manuscript. WWC conceived the study, checked the data and revised the manuscript. All authors have read and approved the final manuscript.

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Abstract

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Background and aims: Visceral adipose tissue has been indicated closely connected with the severity of acute pancreatitis (AP). Visceral adiposity index (VAI) is a mathematical model that consists of waist circumference, body mass index, triglyceride and high-density lipoprotein cholesterol, which has been demonstrated to be a better indicator of visceral fat than other traditional indices. This study aimed to evaluate the relationship between VAI and the severity of AP.

Methods: A retrospective analysis was conducted on a cohort of 1174 patients diagnosed with AP. These patients were categorized into two groups based on their VAI values: the normal VAI (NVAI) group and the elevated VAI (EVAI) group.

Results: The EVAI group were much younger, mainly male and had higher incidence of severe acute pancreatitis (SAP) compared with the NVAI group ($p < 0.001$). The EVAI group developed higher incidences of persistent respiratory failure, acute peripancreatic fluid collection (APFC) and acute necrotic collection (ANC). The VAI level and the percentage of EVAI showed an increasing trend with the severity of AP ($p < 0.001$). EVAI was the most independent risk factor for persistent respiratory failure (OR = 6.405, 95% CI 2.317-17.705), APFC (OR = 2.093, 95% CI 1.255-3.578) and ANC (OR = 4.910, 95% CI 1.736-13.887).

Conclusions: EVAI was strongly related to the severity of AP. It was the most independent risk factor of persistent respiratory failure, APFC and ANC.

Keywords： Acute pancreatitis. Lipid metabolism. Obesity. Visceral adiposity index.

INTRODUCTION

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Acute pancreatitis (AP) is a common acute abdominal disease with increasing incidence year by year (1). Most courses of AP are mild and self-limiting. However, about 15 to 25% of AP patients progress to severe disease with a mortality rate of 36 to 50%, and are usually accompanied by systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (2, 3).

Recent studies have demonstrated that lipid metabolism disorders have an important impact on the severity and clinical outcome of AP, especially obesity (4, 5). A meta-analysis showed that obesity is associated with local complications, organ failure (OF) and high in-hospital mortality in AP patients, which could be used as a prognostic factor (6). Adipose tissue can be divided into white adipose tissue and brown adipose tissue. There are two types of white adipose tissue, subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) respectively (8, 9). Natu et al. reported that VAT was closely associated with severe AP (SAP), necrosis, and multisystem OF (11). Another propensity score matching study of 306 patients found that VAT was an independent predictor of AP (12).

Several clinical measurements have been described to evaluate obesity have been described in previous studies. Of these, body mass index (BMI) fails to differentiate between muscle mass and fat mass, and their distribution. Other indexes, waist circumferenceand waist to height ratio are good indicators of abdominal fat, however, they have limitations in distinguishing SAT from VAT. Visceral adiposity index (VAI) is a mathematical model that consists of BMI, waist circumference, triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C), which has been demonstrated to be a better indicator of VAT than other traditional indices (13, 14).

In 2021, a retrospective study demonstrated that the VAI is a valuable predictor of the severity of hypertriglyceridemia AP (HTG-AP) (15). However, this study was conducted in AP irrespective of etiology. Moreover, although this study explored the correlation between VAI and OF and local complications, it did not further study the specific types of OF and local complications. Based on these, we conducted a larger study with a more extensive sample size to elaborate the value of VAI in the assessment of AP severity irrespective of etiology and find the optimal cut-off points for predicting OF and local complications.

METHODS

Study population and data collection

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In this retrospective study, we included a total of 1174 patients diagnosed with AP from January 1st, 2016 to December 31st, 2020 at the department of gastroenterology of Clinical Medical College of Yangzhou University, China. It was approved by the ethics committee of the hospital on August 4th, 2020 (No. 2020ky-047) and the protocol complied with the ethical guidelines of the 1975 Declaration of Helsinki. This study has been registered in the Chinese Clinical Trial Registry (Registration number: ChiCTR2200055425). We extracted medical history, anthropometric and laboratory data from the hospital information system. The time of blood sample collection and CT examination were within 24 hours after admission. The detailed flow chart is shown in Figure 1.

Definition and classification of acute biliary pancreatitis**, HTG and HTG-AP**

Acute biliary pancreatitiswas defined by either biliary sludge or gallstones on imaging, or a dilated common bile duct on imaging (>8 mm in patients with ≤75 years old or >10 mm in patients with >75 years old), or an increased alanine aminotransferase level of more than twice the upper limit of normal (16). According to the Endocrine Society Clinical Practice Guidelines (17), HTG level was determined above 1.7 mmol/L (17, 18). And HTG-AP is characterized by the clinical syndrome of acute pancreatitis in the presence of either 11.3 mmol/l or 5.65 mmol/l accompanied by milky serum (19). Patients' TG levels were traced to the first detection at the beginning of the onset of AP especially for patients with medical history.

Inclusion and exclusion criteria

Diagnosis criteria for AP required at least two of the following three points: (1) typical upper abdominal pain, (2) the elevation of pancreatic enzyme (serum amylase and/or lipase) levels at least three times above the upper limit of normal, and (3) radiological findings showing characteristic features of AP (20). The severity of AP was defined by the revised Atlanta classification 2012 (21). Only the patients aged 18-80 years old hospitalizing within 7 days after onset of AP were included in the study. Major exclusion criteria were age > 80 or < 18 years, serious comorbidities at admission, such as chronic renal failure, pregnancy, malignant tumor, and those with incomplete data.

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Calculation of BMI, waist circumference **and VAI**

BMI was calculated as the weight (kg) divided by the square of the height (m). Waist circumference was estimated by abdominal CT as following: the navel level on abdominal CT was firstly selected, then the horizontal axis and vertical axis were measured, and the waist circumference was calculated by standard ellipse formula (Ellipse circumference = the coefficient × (short axis + long axis)/2). The VAI for men and women was calculated according to the following formulas: VAI (males) = (waist circumference /(39.68 + (1.88 × BMI))) × (TG/1.03) × (1.31/HDL-C) and VAI (females) = (waist circumference /(36.58 + (1.89 × BMI))) × (TG/0.81) × (1.52/HDL-C). Waist circumference is measured in centimeters (cm), BMI in kg/m^2, TG and HDL-C in mmol/L (22).

Classification of subjects

The critical value of VAI value was 2.30 for men and 3.12 for women, which were calculated according to the pre-determined cut-off points of each parameter: Waist circumference was 90 cm for men and 80 cm for women (23), BMI level was 18.5 kg/m^2 (24), TG concentration was 1.7 mmol/L and HDL-C concentration was 1.0 mmol/L(23). In our study, subjects were divided into two groups according to the critical VAI value. The normal VAI (NVAI) group: VAI < 2.30 for men and < 3.12 for women. The elevated VAI (EVAI) group: VAI ≥ 2.30 for men and ≥ 3.12 for women.

Statistical analyses

Statistical analyses were carried out by SPSS version 25.0 and MedCalc statistical software package version 20.0. The data were expressed as the mean ± standard deviation or median ± interquartile range for continuous variables, and percentages for categorical variables. Student's t test or Mann-Whitney U test was applied for comparisons of continuous data between the two groups. Categorical data were tested by chi-square test or Fisher's exact test. Logistic regression analyses were performed to select independent risk factors associated with OF and local complications of AP patients. And the factors with p value＜0.2 in univariate analysis were then included in multivariate analysis. Receiver operating characteristic (ROC) curves were created to assess the diagnostic strength of VAI. Areas under the curves (AUCs) were calculated to compare the prediction efficiency of the

different indices. The cut-off value of each indicator was determined by the highest value of Youden index score. The results were considered statistically significant when *p* value < 0.05 (two-sided).

RESULTS

Patients' demographic and clinical characteristics

A total of 1174 patients diagnosed with AP were included in this study. The mean age of our patients was 51.4 ± 15.9 years while 61.8% of them were male. The patients in the EVAI group were much younger (45.3 ± 12.9 vs 57.9 ± 16.3) and mainly male (73.4% vs 49.5%) compared to patients in the NVAI group. The causes of AP were gallstone-related in 482 (41.1%) patients, hypertriglyceridemic in 357 (30.4%) patients, idiopathic in 252 (21.5%) patients and alcohol abuse in 43 (3.7%) patients. Among these, HTG was the main etiology in the EVAI group (56.9%), while biliary was the leading cause of NVAI group (61.0%). In addition, BMI and waist circumference were significantly higher in the EVAI group compared to the NVAI group. In terms of biochemical indexes, the WBC and TG concentrations were significantly higher while HDL-C and Ca^{2+} levels were significantly lower in the EVAI group (*p* < 0.001). These results demonstrated that the severity of AP was greater in the EVAI group. Demographic and clinical characteristics of our patients are shown in Table 1.

Comparison of the clinical outcomes between the two groups

Based on the revised Atlanta classification, there were 298 (25.4%) MAP patients, 812 (69.2%) moderately severe acute pancreatitis (MSAP) patients and 64 (5.5%) SAP patients. As shown in Table 2, the incidences of MSAP (75.5% vs 62.4%, *p* < 0.001) and SAP (7.4% vs 3.4%, *p* < 0.001) were significantly higher in the EVAI group compared to those in the NVAI group. In the aspect of OF, the incidence of persistent OFwas significantly higher in the EVAI group, especially for persistent respiratory failure (5.4% vs 2.1%, *p* = 0.003). However, there were no differences in the incidences of persistent heart failure, persistent renal failure and transient OF (*p* > 0.05). Furthermore, higher incidences of acute peripancreatic fluid collection (APFC) and acute necrotic collection (ANC) were found in the EVAI group compared with NVAI group (*p* < 0.05).

Correlation of VAI with the severity of AP

To further explore the relationship between VAI and severity of AP, we compared the VAI levels and the percentage of EVAI in the MAP, MSAP and SAP subgroups. As shown in Table 2, the VAI level showed an increasing trend with the severity of AP (1.64 (0.85-4.98), 3.20 (1.26-16.03), 6.84 (2.24-30.92), $p < 0.001$). Meanwhile, the percentage of EVAI progressively increased in the MAP (34.9%), MSAP (56.5%) and SAP groups (70.3%) ($p < 0.001$).

Logistic regression analysis of persistent respiratory failure in AP patients

Logistic regression analysis was performed to explore the risk factors associated with persistent respiratory failure. Univariate and multivariate logistic analysis indicated that Age > 60 years and EVAI were both independent risk factors for persistent respiratory failure ($p < 0.05$). And EVAI (OR = 6.405, 95% CI 2.317-17.705) was more dangerous than age > 60 years (OR =4.779, 95% CI 2.259-10.111) (Table 3 and Table S1).

Logistic regression analysis of local complications in AP patients

The results of logistic regression analysis among risk factors associated with APFC and ANC are shown in Table 3 and Table S1. Multivariate analysis showed that hypertension (OR =1.603, 95% CI (1.160-2.214)) and EVAI (OR = 2.088, 95% CI (1.222-3.567)) were independent risk factors for APFC ($p < 0.05$), while smoker (OR = 2.376, 95% CI (1.239-4.557)) and EVAI (OR = 4.672, 95% CI (1.646-13.262)) were independent risk factors for ANC ($p < 0.05$). Moreover, EVAI was the most dangerous factor for both APFC and ANC.

DISCUSSION

In this study, patients with higher VAI levels at admission were more likely to be young male and have higher incidences of SAP, persistent respiratory failure, APFC and ANC. EVAI was the most independent risk factor of persistent respiratory failure, APFC and ANC.

VAI includes both anthropometric measures and laboratory indexes that could be better to reflect dyslipidemia and the distribution of abdominal fat, which was proposed by

Amato et al. in 2010(25). It has already been reported to be closely associated with cardiovascular disease, metabolic syndrome and many other diseases (26, 27). A study by Zhou C et al. revealed that VAI was associated with the risk of new-onset type 2 diabetes and new-onset impaired fasting glucose in Chinese hypertensive patients(28). Moreover, a population-based study by He SY et al. showed that VAI was negatively correlated with lung function in the Chinese population (29). A prospective study with 4-year follow-up by Xu CN et al. also reported that the VAI level was an independent risk factor of metabolic dysfunction-associated fatty liver disease (MAFLD) and there was a dose-response relationship between VAI level and incidences of MAFLD (30). To date, only a small sample study explored the relationship between VAI and HTG-AP. There is still a lack of large sample research about VAI in AP patients.

Our study found that higher VAI was more common in young men (45.3 ± 12.9). Previous studies have also suggested similar results between VAI and other diseases. Wen J et al. found that young men had a higher level of VAI in newly diagnosed type 2 diabetes mellitus patients (31). Wang YD et al. found that higher VAI levels were significantly correlated with males in a population-based study (32). Another study by Nam KW et al. showed that VAI had a negative correlation with age in males of neurologically healthy population (33). This may be related to the unhealthy eating habits and lifestyle of young men, such as high-calorie diet, excessive drinking, sedentariness, staying up late and so on, which could easily lead to the accumulation of VAT. In addition, our findings demonstrated that that HTG was the main etiology in EVAI group, which may be related to the lipid metabolism disorders such as dyslipidemia, fatty liver and even fatty pancreas. This finding is in agreement with previous studies that VAI was positively correlated with TG in other diseases (34).

Additionally, the results also clearly showed that higher VAI was associated with a higher incidence of SAP, persistent respiratory failure, APFC and ANC in AP patients. Moreover, logistic regression analysis indicated that EVAI was the independent risk factor of persistent respiratory failure, APFC and ANC, and had the highest OR among all independent risk factors respectively.

Mechanisms by which VAT may exacerbate the severity of AP, especially APFC, ANC and respiratory failure are very complicated. First of all, necrosis of VAT could release a large

amount of unsaturated fatty acids (UFAs), which may result in pancreatic saponification. Severe lipotoxicity of UFAs may induce the OF in AP patients. Second, excess of VAT could result in the decreased level of adiponectin, which not only leads to the increased secretion of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, but also affect the clearance of UFAs and oxidation of fatty acid in mitochondria. That exacerbates the inflammatory response, which may be associated with the occurrence of APFC, ANC and OF (35). Third, excessive VAT could reduce lung compliance and decrease lung volume, thereby impairing lung function. Furthermore, VAT can also affect the mechanical properties of the respiratory system by altering the structure and movement of diaphragm, then eventually exacerbate respiratory failure (29, 32).

We acknowledge that there are several limitations. Firstly, the measurement of patients' waist circumference was conducted using abdominal CT instead of a soft ruler, potentially introducing data bias. Secondly as TG levels decreased rapidly with fasting, the TG levels may have some bias after the admission. The measurement of TG was within 24 hours after admission which may fluctuate with fasting and influence VAI calculations. Thirdly, as this study was conducted in a single center, it is necessary to further validate the findings in multi-center studies. Finally, the participants in our study were all Chinese, more studies are needed to determine whether there was racial bias in our findings.

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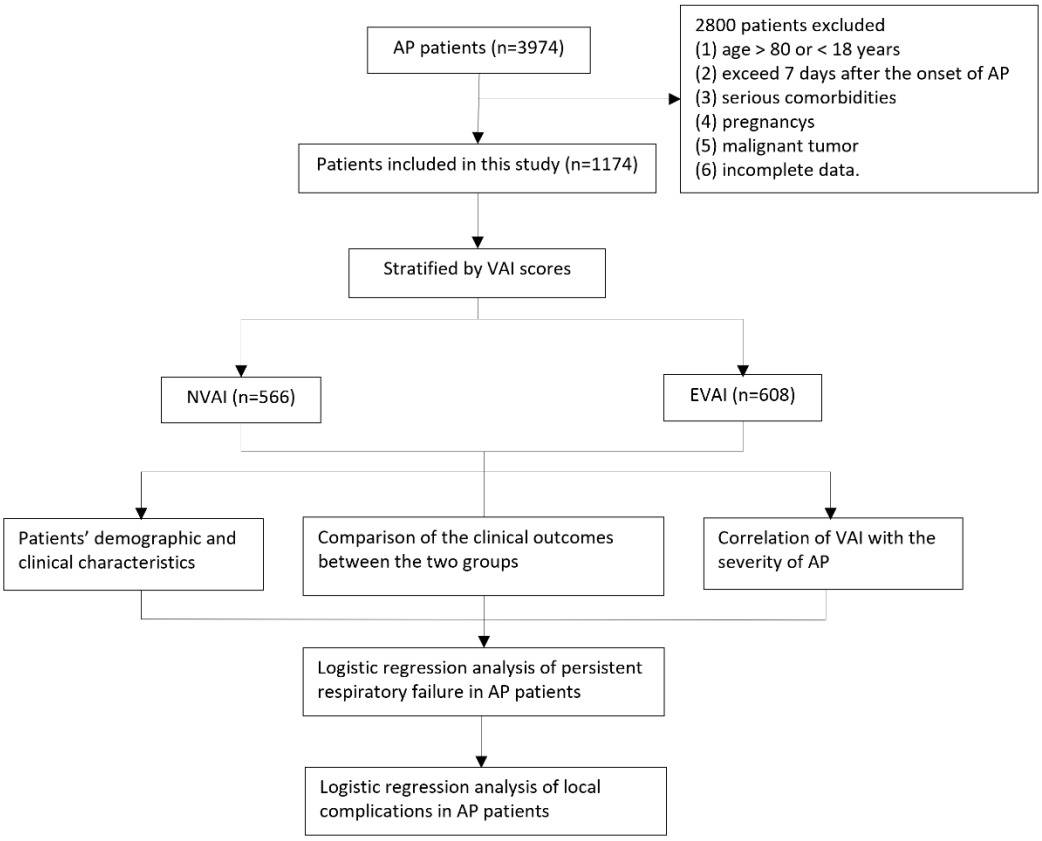

Figure 1. The distribution of AP patients.

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Table 1. Demographic and clinical characteristics of analyzed patients

Variables	Overall (n=1174)	NVAI (n=566)	EVAI (n=608)	*P*
Age (years)	51.4±15.9	57.9±16.3	45.3±12.9	<0.001
Sex (n, %)				<0.001
Male	726 (61.8)	280 (49.5)	446 (73.4)	
Female	448 (38.2)	286 (50.5)	162 (26.6)	
BMI (kg/m2)	25.2±3.8	24.1±3.8	26.1±3.7	<0.001
Waist circumference (cm)	86.7±10.2	83.2±9.2	90.0±10.0	<0.001
Etiology (n, %)				<0.001*
Biliary	482 (41.1)	345 (61.0)	137 (22.5)	<0.001
Hypertriglyceridemia	357 (30.4)	11 (1.9)	346 (56.9)	<0.001
Alcohol	43 (3.7)	21 (3.7)	22 (3.6)	0.933
ERCP	3 (0.3)	2 (0.4)	1 (0.2)	0.612
Idiopathic	252 (21.5)	166 (29.3)	86 (14.1)	<0.001
Mixedness	19 (1.6)	7 (1.2)	12 (2.0)	0.317
Others	18 (1.5)	14 (2.5)	4 (0.7)	<0.001
Laboratory indicators				
TG (mmol/L)	1.7 (0.9-6.2)	0.9 (0.6-1.1)	5.7 (2.7-17.5)	<0.001
LDH (IU/L)	262.0 (201.0-432.0)	257.0 (198.0-435.0)	267.5 (204.8-431.5)	0.410
HDL-C (mmol/L)	1.0 (0.7-1.3)	1.2 (1.0-1.5)	0.8 (0.6-1.0)	<0.001
WBC (10^9/L)	11.5 (8.6-14.8)	10.7 (7.8-13.9)	12.4 (9.5-15.3)	<0.001
BUN (mmol/L)	5.1 (3.9-6.6)	5.2 (3.9-6.7)	4.9 (3.8-6.4)	0.138
CRE (umol/L)	73 (61-87)	72 (61-84)	74 (61-89)	0.153
Ca^{2+} (mmol/L)	2.2 (2.1-2.3)	2.2 (2.1-2.3)	2.2 (2.0-2.3)	<0.001

Abbreviations: BMI, Body Mass Index; ERCP, Endoscopic Retrograde Cholangiopancreatography; TG, triglyceride; LDH, lactate dehydrogenase; HDL-C, High-density lipoprotein cholesterol; WBC, White blood cells; BUN, Blood urea nitrogen; CRE, Creatinine.

* Fisher's exact test.

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Table 2. The relationship between VAI and severity of AP

Comparison of the clinical outcomes between the two groups				
Variables	Overall	NVAI	EVAI	*P*
Atlanta classification (n, %)				<0.001
MAP	298 (25.4)	194 (34.3)	104 (17.1)	<0.001
MSAP	812 (69.2)	353 (62.4)	459 (75.5)	<0.001
SAP	64 (5.5)	19 (3.4)	45 (7.4)	<0.001
Persistent OF	61 (5.2)	19 (3.4)	42 (6.9)	0.006
Persistent heart failure	8 (0.7)	5 (0.9)	3 (0.5)	0.493
Persistent respiratory failure	45 (3.8)	12 (2.1)	33 (5.4)	0.003
Persistent renal failure	25 (2.1)	9 (1.6)	16 (2.6)	0.217
Transient OF	35 (3.0)	21 (3.7)	14 (2.3)	0.156
Transient heart failure	18 (1.5)	11 (1.9)	7 (1.2)	0.270
Transient respiratory failure	17 (1.4)	9 (1.6)	8 (1.3)	0.694
Transient renal failure	9 (0.8)	2 (0.4)	7 (1.2)	0.181
Local complications (n, %)	868 (73.9)	367 (64.8)	501 (82.4)	<0.001
APFC (n, %)	863 (73.5)	364 (64.3)	499 (82.1)	<0.001
ANC (n, %)	43 (3.7)	12 (2.1)	31 (5.1)	0.007
PPC (n, %)	20 (1.7)	8 (1.4)	12 (2.0)	0.459
WON (n, %)	4 (0.3)	0 (0.0)	4 (0.7)	0.125
IPN (n, %)	3 (0.3)	1 (0.2)	2 (0.3)	0.100
Correlation of VAI with the severity of AP				
Variables	MAP	MSAP	SAP	*P*
VAI	1.64(0.85-4.98)	3.20(1.26-16.03)	6.84(2.24-30.92)	<0.001
EVAI%	104 (34.9)	459 (56.5)	45 (70.3)	<0.001

Abbreviations: OF, organ failure; POF, persistent organ failure; APFC, acute peripancreatic fluid collection; ANC, acute necrotic collection; PPC, pancreatic pseudocyst; WON, walled-off necrosis; IPN, infectious pancreatic necrosis.

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Table 3. Multivariate analysis of complications in AP patients

Persistent respiratory failure		
Variable	OR (95%CI)	*P*
Age>60	4.779 (2.259-10.111)	<0.001
HTG	0.781 (0.287-2.129)	0.629
Recurrence ≥2 times	2.088 (0.925-4.716)	0.077
Hypertension	1.130 (0.579-2.204)	0.721
Diabetes	0.080 (0.521-2.242)	0.836
EVAI	6.405 (2.317-17.705)	<0.001
APFC		
	OR (95%CI)	*P*
Male	0.956 (0.720-1.269)	0.755
HTG	1.213 (0.708-2.078)	0.483
Hypertension	1.603(1.160-2.214)	0.004
Diabetes	1.426 (0.951-2.140)	0.086
Coronary heart disease	1.698 (0.465-6.193)	0.423
Smoker	0.781 (0.561-1.086)	0.142
EVAI	2.088 (1.222-3.567)	0.007
ANC		
	OR (95%CI)	*P*
Male	1.357 (0.671-2.743)	0.396
HTG	0.435 (0.166-1.142)	0.091
Hypertension	1.579 (0.832-2.995)	0.162
Smoker	2.376 (1.239-4.557)	0.009
EVAI	4.672 (1.646-13.262)	0.004