Clinical and Experimental HEPATOLOGY

Clin Exp HEPATOL 2021; 7, 3: 264–269
DOI: https://doi.org/10.5114/ceh.2021.109345
Received: 24.12.2020, **Accepted:** 7.04.2021, **Published:** 11.10.2021

Original paper

Effect of abdominal fat distribution on severity of acute pancreatitis

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Abstract

Aim of the study: Obesity is a well-determined risk factor for acute pancreatitis. Increased visceral fat has been shown to increase the proinflammatory environment experienced by patients. In this study, we aimed to research the correlation between abdominal fat distribution parameters measured with computed tomography (CT) and severity of acute pancreatitis (AP).

Material and methods: The study included patients monitored due to AP in the internal medicine clinic of GOP Education and Research Hospital from January 2015 to December 2018. The Acute Physiology and Chronic Health Evaluation (APACHE) score, the Imrie score and the Bedside Index of Severity in Acute Pancreatitis (BISAP) scores were calculated. Advanced image processing analysis software (INFINIT Xelis, v 1.0.6.3) was used to calculate individual abdominal fat distribution parameters from CT screening with division of abdominal tissues. Measurements were performed from −50 to −250 Hounsfield units (HU) between vertebrae L2-L3.

Results: When mild and moderate AP groups were compared, there were statistically significant differences in duration of hospital stay and scoring (APACHE, Imrie and BISAP) ($p < 0.001$), while there were no significant differences in abdominal fat distribution parameters ($p > 0.05$). There was no significant correlation of visceral and subcutaneous fat volumes with development of systemic complications, while a significant correlation was identified for visceral to total fat tissue area ratio (VTR) with local complications ($p < 0.001$). Pearson correlation analysis found no correlations of mortality and pancreatitis severity with visceral (VFA) and subcutaneous fat area (SFA) ($p > 0.05$). Positive correlations were identified for VFA with Imrie, BISAP and APACHE scores ($p < 0.01$), and positive correlations were identified for visceral adipose tissue (VAT) with visceral to subcutaneous fat ratio (VSR) and APACHE scores ($r = 0.256$ and 0.252, respectively, $p < 0.001$). Positive correlations were identified for VTR and VSR ratios with BISAP scores ($r = 0.266$ and $r = 0.277$, respectively, $p < 0.001$).

Conclusions: In patients with AP diagnosis and abdominal CT scans, increased VFA and VTR ratio were found to be associated with increased AP clinical scores with no significant correlation identified in terms of local/systemic complication development. Our study shows that VFA is linked to AP clinical scoring systems and should be included in AP predictive scoring systems.

Key words: acute pancreatitis, emergency service, scoring system, severe acute pancreatitis.

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Introduction

Acute pancreatitis (AP) is suddenly developing inflammation of the pancreas and is the gastrointestinal system disease requiring most frequent admission to intensive care from the emergency service, with increasing incidence worldwide [1-3]. The increase in AP incidence involves many factors such as obesity, alcohol, gallstones, medications, infection and trauma [4]. Obesity is a well-defined risk factor for acute pancreatitis. Increased visceral fat was shown to increase the proinflammatory environment experienced by patients.

In recent years, new scoring systems have been created, e.g. the Bedside Index Severity Acute Pancreatitis (BISAP), the Glasgow Prognostic Score (Imrie) and the Computed Tomography Severity Index (CTSI-Balthazar), for more appropriate determination of AP severity under emergency service conditions [5-7]. In this study, we aimed to research the correlation between abdominal fat distribution parameters measured with computed tomography (CT) and severity of acute pancreatitis.

Material and methods

Study population

The study included patients monitored for acute pancreatitis from January 2015 to December 2018. Data were retrospectively investigated. All patients had Acute Physiology and Chronic Health Evaluation (APACHE), Imrie and BISAP scores calculated within the first 24 hours of admission. The study included patients with AP diagnosis aged over 18 years. From these patients, those with traumatic pancreatitis diagnosis and pregnant cases were excluded from the study. Patient age, gender, height, weight, comorbid diseases and medications used were recorded. Patients had demographic characteristics, laboratory results, pulmonary radiography and abdominal CT results evaluated and BISAP, Imrie and Balthazar scores calculated [8, 9].

Definition of acute pancreatitis severity

Patients with AP diagnosis were classified as having mild, moderate or severe AP (SAP) according to the revised Atlanta criteria from 2012 along with AP clinical, radiological and anatomical findings [10]. Cases of patients with organ failure within 48 hours after attendance at hospital and/or dead patients were considered as severe AP [11]. Additionally, cases of patients without organ failure but with development of local severe complications such as necrosis or abscess were considered as severe AP [10]. Organ failure status was calculated using the Marshall score. In this scoring, if patients receive ≥ 2 points, they are accepted as having developed organ failure [12]. All patients with AP diagnosis were monitored and treated according to the evidence-based AP management guidelines of the International Association of Pancreatology and American Pancreatic Association (IAP/APA) [13].

Assessment of abdominal fat tissue distribution with CT: With division of abdominal tissue, advanced image processing analysis software (INFINIT Xelis, v 1.0.6.3) was used to calculate individual abdominal fat distribution parameters from CT screening with measurements performed from –50 to –250 HU between vertebrae L2-L3.

Power analysis

A statistical power analysis was performed for sample size estimation, based on data from the published study by Natu *et al.* (N = 252) [14]. The effect size in this study was 0.3, considered to be extremely large using Cohen's (1988) criteria. With an $\alpha = 0.05$ and power = 0.80, the projected sample size needed with this effect size (GPower 3.1.9.4 software) is approximately N = 64+ for this simplest between/within group comparison. Thus, our proposed sample size of n = 170 will be more than adequate for the main objective of this study and should also allow for expected attrition and our additional objectives of controlling for possible subgroup analysis.

Statistical analyses

Analysis of data used the program SPSS Statistics 21.0 (IBM Corporation, Armonk, New York, USA). Since there were no patients suitable for the diagnosis of severe pancreatitis in our study, the patients were grouped as mild and moderate AP. Normal distribution of data was checked with the Kolmogorov-Smirnov test. Descriptive statistics are given as mean ± standard deviation (SD), while nominal variables are given as case numbers and percentages (%). Continuous variables with normal distribution were compared with the Student t test, while continuous variables without normal distribution were compared with the Mann-Whitney U test. The χ^2 or Fisher's exact test was used to compare categoric variables. The Mann-Whitney U test was used with Monte Carlo simulation technique to compare two independent groups with each other according to quantitative data. Spearman's rho test was used to examine the correlations of variables with each other. Correlation between APACHE, Imrie, and Balthazar scores and abdominal fat distributions was made with a bipartial correlation analysis. The statistical significance level for the obtained data was interpreted with the p value. $P < 0.05$ was accepted as statistically significant.

Results

The mean age in the study was 58.7 ±18.3 years, with a total of 174 patients, 107 females (61.5%) and 67 males (38.5%). Of patients included in the study, 142 (81.6%) had mild and 32 (19.4%) had moderate AP.

Among all patients, 114 (65.5%) were identified to have biliary pancreatitis, and this was identified more frequently in patients with moderate AP ($p < 0.001$) (Table 1). Moderate AP patients had longer duration of hospital stay, and higher Imrie and Balthazar scores ($p < 0.001$). Additionally, no significant differences were identified between the groups in terms of Apache and BISAP scores (Table 1). When the groups were assessed in terms of visceral fat tissue, subcutaneous fat tissue and total fat tissue ratios, no significant difference was identified ($p > 0.05$).

When patients are assessed in terms of complication development, 2 out of 11 patients developing systemic complications were identified to develop simultaneous local complications. Patients developing systemic complications were found to be older and have higher BISAP scores (Table 2). Patients developing local complications had a higher Balthazar score than those who did not develop local complications ($p < 0.001$) (Table 2). No significant differences were identified between patients developing and not developing complications in terms of visceral adipose tissue (VAT) volume, subcutaneous fat tissue area (SFA) and total fat tissue area (TFA) measurements, visceral to subcutaneous fat tissue ratio (VSR), visceral to total fat tissue area ratio (VTR) or subcutaneous to total fat tissue area ratio (STR).

In our study, a weak positive correlation was identified for VAT and VSR with APACHE scores ($r = 0.256$ and $r = 0.252$, respectively, $p < 0.001$) (Table 3). A weak positive correlation was identified for VTR and VSR ratios with BISAP scores ($r = 0.266$ and $r = 0.277$, respectively, $p < 0.001$) (Table 3). No significant correlation was identified between clinical scoring systems and pancreatitis severity.

Discussion

Obesity is a well-determined risk factor for AP. Increased visceral fat was shown to increase the proinflammatory environment experienced by patients. In our study, we did not identify a correlation of visceral fat tissue surface area or any abdominal fat tissue area ratios, measured with CT and assessed as an objective measure of obesity, with the severity of AP.

Acute pancreatitis may have variable clinical status from a mild degree of inflammation of the pancreas to severe progressive pancreatic necrosis. In 80% of patients the clinical course is self-limiting and they improve without recurrence. For nearly 15% of patients, pancreatic necrosis requiring a few weeks of hospital

Table 1. Clinical and laboratory characteristics of patients

Variable	Mild AP (n = 142) Mean ±SD	Moderate AP (n = 32) Mean ±SD	P value
Age (years)	58.7 ±18.41	58.03 ±17.73	0.855
Gender (M/F)	90/52	17/15	0.101
Etiology (biliary/nonbiliary)	87/55	27/5	**< 0.001**
Mortality	None	3	**< 0.001**
APACHE score	4.13 ±2.95	4.77 ±3.20	0.295
	Median (IQR)	**Median (min./max.)**	**P value**
Hospitalization delay (days)	5 (4)	7 (10)	**< 0.001**
Imrie score	1 (2)	2 (2)	**< 0.001**
BISAP score	1 (1)	1 (2)	**0.007**
Balthazar score	1 (2)	3 (2)	**< 0.001**
VAT (cm^2)	142.83 (130.27)	146.80 (137.06)	0.178
SAT (cm^2)	169.79 (134.07)	132.99 (117.24)	0.166
Total fat area (cm^2)	327.39 (203.54)	292.11 (171.55)	0.099
VSR	0.840 (0.489)	0.978 (0.772)	0.863
VTR	0.460 (0.215)	0.494 (0.188)	0.961
STR	0.549 (0.211)	0.506 (0.188)	0.961

Data are shown as mean ±SD; Wilcoxon signed ranks test (Monte Carlo);
VAT – visceral tissue total surface area, SAT – subcutaneous tissue total surface area, VSR – visceral to subcutaneous fat area ratio, VTR – visceral to total fat area ratio, STR – subcutaneous to total fat area ratio, BISAP – Bedside Index Severity Acute Pancreatitis score; Imrie, Glasgow Prognostic Score

Table 2. Comparison of groups with absence of systemic and local complications

Variable	Systemic complications		*P* value	Local complications		*P* value
	Present (*n* = 11) Mean ±SD	Absent (*n* = 163) Mean ±SD		Present (*n* = 10) Mean ±SD	Absent (*n* = 164) Mean ±SD	
Age (years)	77.64 ±13.95	57.29 ±17.80	**< 0.001**	59.30 ±15.37	58.54 ±18.44	0.902
APACHE score	7.27 ±2.37	4.04 ±2.92	0.001	6.10 ±4.06	4.13 ±2.89	0.167
Imrie score	3.09 ±1.51	1.17 ±1.05	0	2.3 ±1.49	1.23 ±1.13	0.053
	Median (IQR)	**Median (IQR)**	***P* value**	**Median (IQR)**	**Median (IQR)**	***P* value**
Hospitalization delay (days)	10 (14)	5 (4)	0.11	17 (8)	5 (3)	**0.04**[t]
BISAP score	2 (1)	1 (1)	**< 0.001**	1 (2)	1 (1)	0.217
Balthazar score	3 (4)	2 (3)	0.202	4 (1)	2 (3)	**< 0.001**[t]
VFA (cm^2)	105.18 (78.24)	144.01 (129.43)	0.397	213.06 (112.14)	141.81 (128.59)	0.842
SFA (cm^2)	112.36 (117.93)	166.47 (129.16)	0.294	123.57 (94.25)	166.47 (132.71)	0.021
TFA (cm^2)	241.47 (211.57)	327.31 (184.89)	0.108	341.98 (183.37)	326.40 (199.74)	0.132
VSR	0.874 (0.276)	0.882 (0.777)	**0.037**	1.663 (2.073)	0.858 (0.720)	0.066
VTR	0.466 (0.820)	0.473 (0.217)	**0.007**	0.610 (0.303)	0.464 (0.199)	0.60
STR	0.534 (0.082)	0.536 (0.212)	**0.007**	0.390 (0.303)	0.540 (0.197)	0.60

Data are shown as mean ±SD.

[t]Independent samples t test (bootstrap).

VFA – visceral tissue total surface area, SFA – subcutaneous tissue total surface area, total surface area, VSR – visceral to subcutaneous fat area ratio, VTR – visceral to total fat area ratio, STR – subcutaneous to total fat area ratio, BISAP – Bedside Index Severity Acute Pancreatitis score, Imrie, Glasgow Prognostic Score

Table 3. Correlations between acute pancreatitis severity scores and abdominal fat tissue measurements

Variable	AP severity scores					
	APACHE		Imrie		BISAP	
	r	*p*	*r*	*p*	*r*	*p*
VFA	0.256	**< 0.001**	0.271	**< 0.001**	0.199	0.009
SFA	0.011	0.889	0.011	0.889	0.094	0.221
VTR	0.250	**< 0.001**	0.112	0.144	0.266	**< 0.001**
STR	0.230	0.002	0.204	0.008	0.261	**< 0.001**
VSR	0.252	**< 0.001**	0.226	0.003	0.277	**< 0.001**

Partial correlation test, r – correlation coefficient

AP – acute pancreatitis, VFA – visceral tissue total surface area, SFA – subcutaneous tissue total surface area, VSR – visceral to subcutaneous fat area ratio, VTR – visceral to total fat area ratio, STR – subcutaneous to total fat area ratio, BISAP – Bedside Index Severity Acute Pancreatitis score, Imrie, Glasgow Prognostic Score

stay. Of patients developing necrosis, nearly 35% develop multiorgan failure while 20% develop infected pancreatic necrosis. Developing multiorgan failure and infected pancreatic necrosis are associated with mortality rates of 6-23% [15, 16].

Early diagnosis of severe AP and identification of patients requiring aggressive treatment in the early period have vital importance. The Atlanta criteria have been used to determine AP severity and treatment planning since 1992. The IAP/APA diagnostic and treatment guidelines recommend initial hemodynamic status assessment and stabilization of AP patients in the emergency service [13]. In the second stage, it is recommended that patients be assessed with an appropriate scoring system without losing time [17]. There are many scoring systems, e.g. Ranson, APACHE II, BISAP, Imrie, Balthazar, and PANC3, to determine the severity of AP [9, 10, 18].

In the literature, there are many studies investigating the correlation between age and AP mortality. In spite of a low rate of increase in the incidence of AP in recent years, the incidence of severe AP has increased at a relatively high rate. The most important reason for this increase was shown to be the increase in mean patient age [19, 20]. A study by Gardner *et al.* of 70 AP patients found that the mortality rate was significantly

high among patients over 70 years of age (21.4% vs. 7.1%) (OR = 3, $p = 0.028$) [21]. A retrospective study by Wang *et al*. found being ≥ 72 years old was predictive for organ failure in 393 AP patients [22]. In our study, the mean age of patients with AP diagnosis was 58.7 ±18.3 years, and it is considered important that the age variable does not affect the correlation between obesity and pancreatitis, which we aimed to assess in this study.

Another explanation for the relationship between abdominal obesity and the development of severe AP is that obese patients have a chronic proinflammatory state that may predispose to a greater inflammatory response when AP occurs. The evidence supporting this inflammatory response hypothesis comes from two different studies showing increased levels of C-reactive protein and/or proinflammatory cytokines (IL-1β, IL-6 and IL-8) in obese patients with AP compared to non-obese patients [23, 24].

A meta-analysis of five studies showed that obese patients had a 2.9-fold (OR: 2.9, 95% CI: 1.8-4.6) increased risk for severe AP and a 2.1-fold (OR: 2.1, 95% CI: 1.0-4.8) increased risk for AP with a fatal outcome [23].

There is a strong correlation between AP severity and obesity [25, 26]. A study by Katuchova *et al*. reported that patients with body mass index (BMI) > 30 kg/m^2 had more local and systemic complications and that obesity was a significant negative predictor for mortality [27]. Taguchi *et al*. emphasized that like those who are overweight, very thin people were at risk in terms of acute pancreatitis [28]. A study by Krishna *et al*. found that morbidly obese AP patients spent longer in hospital; these patients developed more organ failure and had higher mortality (OR: 1.6, 95% CI: 1.3-1.9) [29]. The reason for this is thought to be that patients with high BMI have an increased pancreatic fat amount which is associated with a direct toxic effect on pancreas parenchyma and delayed healing [26, 27]. Proinflammatory cytokines released in excess amounts from fat tissue are associated with widespread inflammation, which is encountered as another negative factor. In recent studies, many studies have aimed to define the abdominal fat distribution properties of obese people (visceral and subcutaneous).

Sempere *et al*. [30] evaluated waist circumference (WC) and BMI as markers of abdominal fat distribution and found that increased WC (RR: 4.65, 95% CI: 1.17-18.5) and BMI (RR: 2.06, 95% CI: 0.99-4.19) increased the risk of severe AP development. Based on this evidence, abdominal obesity appears to be more relevant in predicting severe AP than general obesity. A study in Japan identified 85 cm waist circumference for men and 90 cm waist circumference for women as equivalent to a 100 cm^2 VFA cut-off point as a marker of disorder risk associated with obesity [31]. Similarly, a Japanese study including 12,443 subjects in visceral fat accumulation and coronary artery disease research (VACATION-J) found that major cardiovascular events were associated with increased VFA; however, no correlation with SFA was found.

A 62-patient study by O'Leary *et al*. [32] showed that increased abdominal VFA level measured with abdominal CT was associated with development of severe AP and increased risk of systemic complications. A study published by Xie *et al*. in 2019 revealed that increased abdominal VFA level and VTR were closely associated with worsening AP [33]. Similarly, in our study, the increases in VFA level and VTR were associated with high AP clinical scores. Additionally, in our study the measurements for fat tissue area were found to be higher than values in the literature. This situation is considered to be associated with the fact that the majority of the mentioned studies concerned East Asian populations. Similar studies performed in our country will reveal the abdominal fat distribution features of people living in this region.

Limitations

This study has important limitations. The first is that our study is a single-center study. Our study was performed in a single province at the only university hospital in the provincial center, which significantly reduces the generalizability of the study. Since the inclusion of patients with a diagnosis of severe AP that we planned before our study could not be achieved, we could only evaluate mild and moderate AP patients. Additionally, fat distribution measurements were retrospectively made on noncontrast enhanced abdomen CT taken for AP diagnosis for patients in the emergency service, and we may not have accessed optimal measurement results due to differences in technique. Due to our study being retrospective, BMI measurements of patients could not be recorded and patients could not be grouped.

Conclusions

In conclusion, patients with AP diagnosis and abdominal CT with increased VFA and VTR had increased mean AP clinical scores, with no significant correlation identified in terms of local/systemic complication development. There is a need for studies on this topic with larger patient groups assessing the abdominal fat distribution in groups comprising patients with different BMI values. Abdominal CT measure-

ment is a useful, repeatable method providing objective results. We believe this method will provide significant contributions to developments in AP diagnosis and treatment.

Disclosure

The authors declare no conflict of interest.

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