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Visceral Abdominal Fat and Muscle Mass Impact on Patients with Severe Acute Pancreatitis' Risk for Death

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Abstract

Aim: The purpose of this study was to look into any correlations between muscle and adipose features and the severity and prognosis of patients hospitalized with Acute Pancreatitis (AP).

Methods: In total, 392 hospitalized patients and 309 controls were enrolled in the study analysis between May 2017, and January 1st, 2022. A computed tomography scan was used for each population to evaluate muscle and adipose parameters. The influence of variables on the development of either Moderately Severe Acute Pancreatitis (MSAP) or Severe Acute Pancreatitis (SAP) was evaluated using univariate and multivariate logistic regression analysis. Cox regression analysis was used to look at the relationships between disease recurrence and death.

Results: The controls had lower levels of Skeletal Muscle Attenuation (SMA) and visceral and subcutaneous adipose tissue attenuation (both $p<0.05$). The AP patients, on the other hand, had higher visceral and subcutaneous adipose tissue (144.25 *vs.* 97.81 cm^2, $p<0.001$). Visceral Adipose Tissue (VAT) and SMA showed a significant difference in the AP severity groups, with p-values of 0.014 and 0.003, respectively. When looking at many factors at once, VAT and SMA were linked to either SAP or MSAP (95% CI 0.953-0.993, p=0.010) or 1.000-1.006, p=0.041, with odds ratios of 1.003 and 0.973, respectively. Low SMA was linked to a higher death rate in MSAP and SAP patients, according to Cox regression analysis (HR: 10.500, 95% CI 1.344-82.025, p=0.025). There was also a link between a lower risk of acute pancreatitis happening again in one year and a VAT loss of more than 17% (HR: 0.427, 95% CI 0.189-0.967, p=0.041).

Conclusion: SMA and VAT significantly impacted the severity and prognosis of AP patients. Patients should maintain a good diet and exercise routine after being released from their VAT and gaining muscle. Their prognosis will improve as a result.

Keywords: Skeletal muscle; Subcutaneous adipose tissue; Recurrent acute pancreatitis; VAT; SMA

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Abbreviations

HU: Hounsfield Units; OR: Odd Ratio; CI: Confidence Interval; L3-SMI: Skeletal Muscle Attenuation; SATA: Visceral Adipose Tissue Attenuation; SAT: Subcutaneous Adipose Tissue

Important Synopsis

According to established information, acute pancreatitis is one of the most common gastrointestinal conditions that cause pain for patients and financial strain on healthcare systems. Research on the relationship between obesity and acute pancreatitis severity has produced inconsistent findings.

Recent Discoveries

In this large European cohort, muscle mass and muscle attenuation were important factors for acute pancreatitis. However, visceral fat parameters and AP severity of acute pancreatitis were not linked in the multivariate analysis, and none of the body parameters could be used to predict disease severity.

Simple Synopsis in Language

Everyone knows that the incidence, recurrence, and mortality of acute pancreatitis are rising yearly. Since Computed Tomography (CT) imaging can accurately estimate skeletal muscle and

abdominal adipose tissue parameters, it is regarded as the gold standard for assessing body composition. Three hundred ninety-two patients with acute pancreatitis and 309 control participants provided us with their CT scans. Patients with acute pancreatitis showed lower Skeletal Muscle Attenuation (SMA) and increased Visceral Adipose Tissue (VAT) compared to the control groups. Elevated VAT is linked to Moderately Severe Acute Pancreatitis (MSAP) and Severe Acute Pancreatitis (SAP). Strong SMA, on the other hand, acted as a buffer for SAP or MSAP. Furthermore, we found a significant correlation between low SMA and a greater death rate in individuals with SAP and MSAP. VAT losses larger than 17 percent were associated with a one-year reduction in the recurrence of acute pancreatitis. Patients should, in our opinion, get education after their hospital stay. A healthy diet and regular exercise can improve a patient's prognosis and quality of life while lowering VAT and enhancing muscular function.

Introduction

Recently, Acute Pancreatitis (AP) has become increasingly common [1]. Patients with Severe AP (SAP) continue to have a high mortality rate despite significant demographic research from the United States, indicating that AP mortality rates decreased from 12% to 2% between 1988 and 2003 [1]. Approximately 15% to 25% of AP patients progress to significant SAP2, and a 2011 survey conducted across Japan revealed that 9.5% of SAP patients died [2,3].

Obesity is linked to unfavorable changes in adipose tissue, resulting in chronic inflammation and other disorders [4]. Proinflammatory cytokines can be effectively produced by adipose tissue [5]. Severe Systemic Inflammatory Response Syndrome (SIRS), which is hypothesized to induce organ dysfunction and result in death in patients with SAP, is facilitated by adipose tissue-derived cytokines. Sarcopenia is associated with poor prognosis, higher risk of complications, and morbidity in cancer patients, regardless of the patient's Body Mass Index (BMI) [6-9]. Only a few studies have been published on AP; hence, the impact of muscle metrics, including Muscle Mass (MM), Mean Muscle Attenuation (MMA), and Visceral Muscle Ratio (VMR) on disease outcome has not been fully examined [10-18].

To begin with, Hypertriglyceridemia, gallstones, alcohol consumption, and other uncommon etiologies frequently contribute to Acute Pancreatitis (AP), a dangerous aseptic inflammatory illness of the pancreas. Worldwide, there are 34 occurrences of acute pancreatitis for every 100,000 people, and the number of hospitalizations has risen rapidly in recent years [4,5]. Most patients hospitalized for Acute Pancreatitis (AP) recover totally. However, it is noteworthy that after healing fully or almost fully, 17% to 29% of patients acquire Recurrent Acute Pancreatitis (RAP), and 3% to 14% of these patients develop Chronic Pancreatitis (CP) [19]. Recent data indicate that patients with Moderately Severe Acute Pancreatitis (MSAP) had a death rate of approximately 2%, whereas patients with Severe Acute Pancreatitis (SAP) had a much higher rate (36%-50%) [5,6]. The grim prognosis of AP has a significant impact on the healthcare system. Recurrent AP has been demonstrated to reduce physical and mental quality of life even in the absence of CP [6]. Computed Tomography (CT) imaging is the gold standard for determining body composition because it can accurately quantify characteristics related to skeletal muscle and adipose tissue [2-8]. Almost all AP patients had abdominal CT scans. When evaluating the degree of AP using clinical scoring systems and abdominal CT at admission, similar outcomes were seen [9]. Although visceral adipose tissue is not a standalone risk factor for AP, a recent systematic study showed a high connection between it and the severity of AP. On the other hand, Visceral Adipose Tissue (VAT) is not a risk factor for SAP; rather, poor Skeletal Muscle Attenuation (SMA) [10]. Recent studies have demonstrated a considerable rise in-hospital mortality in patients with necrotizing pancreatitis when looking at prognostic analyses [11]. A lower overall survival rate was substantially correlated with a lower SMA. Research on the causes of recurrent AP is hard to find in the literature, especially from Egypt countries. The primary objective of our current investigation was to determine whether body composition measurements affected the occurrence of SAP or MSAP. Notably, we looked into the potential connection between people who experience recurrent AP and a decline in body composition. A secondary objective was to assess their impact on the short-term mortality of individuals with SAP and MSAP.

Methods

Analyze the population

For this retrospective study, we collected 392 hospitalized patients with acute pancreatitis and 303 at Zagazig University Hospital between May 1st, 2017, and January 1st, 2022. Three criteria were met for the diagnosis of Acute Pancreatitis (AP): 1) serum lipase or amylase levels three times higher than the upper limit of normal; 2) acute and persistent upper abdominal pain that often radiates to the back; and 3) abdominal CT scans consistent with the appearance of acute pancreatitis [12]. The Atlanta Classification 2013 revision's numerous clinical symptoms and prognoses were used to divide the severity of AP into three categories: Mild, moderately severe, and severe. There were no problems, either local or systemic, or organ failure in mild AP patients. Very few people passed away from MAP. Individuals with moderately severe AP either had transient organ failure that went away in 48 h or endured systemic or local issues. Severe acute pneumonia was defined as ongoing organ failure that persisted for more than 48 h. Individuals suffering from any of the following disorders were unable to take part in the study: (1) cancer, (2) hepatitis, liver cirrhosis, and other severe liver illnesses, and (3) chronic pancreatitis. The only conditions observed in 303 controls were simple gastrointestinal polyps, mild gastroesophageal reflux syndrome, chronic gastritis, or dyspepsia. Cancerous diseases; endocrine and metabolic syndrome, including diabetes mellitus and thyroid dysfunction; (4) several serious illnesses, such as kidney, circulatory, and respiratory failure; (5) autoimmune diseases; and (6) several chronic illnesses, such as liver 392, chronic hepatitis, and liver cirrhosis. These conditions had to be met for the patient to be automatically ruled out. For patients who visited the hospital more than once throughout the survey period due to recurrent AP, the initial recurrence was considered for further analysis. Mortality was computed using clinical case records. The duration of death, expressed in days, between the date of the CT scan and the official date of death.

Examining CT scan results

CT scans of the abdomen were done on every research participant, including controls. All CT images were obtained via the PACS system and measured in DICOM format. CT scans were chosen at the middle level of the third lumbar vertebrae and measured using slice Omatic software (version 5.0; Tomovision, Magog, Canada) to quantify bodily tissues [13]. Various tissues are separated based on varying density thresholds: Subcutaneous Adipose Tissue (SAT) is at −190 to

−30 Hounsfield Units (HU), VAT is at −150 to −50 HU, and Skeletal Muscular Tissue (SMT) is from −29 to +150 HU. After that, a skilled operator painstakingly sketches out each tissue. Figure 1 shows skeletal muscle in red, visceral adipose tissue in faint yellow, and subcutaneous adipose tissue in shinning green. The areas and mean attenuations (HU) of various tissues were automatically calculated. The cross-sectional skeletal muscle area was scaled to height (cm^2/m^2) to compute the Skeletal Muscle Index (SMI). Skeletal muscle Attenuation (SMA), computed using the mean Hounsfield Unit (HU) value of every pixel in SMT, was correlated with the triglyceride content of the muscle [14] (Figure 1). Body composition in many AP patients is displayed by computed tomography images of differing degrees of severity. Figure 1A-1C show mild, moderately severe, and severe AP, respectively. Skeletal muscle is represented by red, visceral adipose tissue is represented by yellow, and subcutaneous adipose tissue is represented by green. VAT increased gradually at the same rate as the degree of AP (54.01 cm^2, 135.8 cm^2, and 372.4 cm^2). Conversely, SMA (52.41 HU, 37.08 HU, and 18.99 HU) showed a tendency toward decline.

SAP treatment

When we identified a patient with SAP, we began providing assistance in the Intensive Care Unit (ICU), following Japanese regulations [9]. We observed diastolic blood pressure maintained at 65 mmHg or more and the output of urine remained at 0.5 ml/kg/h or more within 48 h of commencement, to put it briefly. Fentanyl, a wide-spectrum antimicrobial agent used as a preventative agent within 72 h of initiation, and a protease inhibitor were used to treat the pain. Acute Necrotic Collection (ANC), Acute Pancreatic Fluid Collection (APFC), Pancreatic Pseudocysts (PPC), and Walled-Off Necrosis (WON) with or without infection are local consequences. Precautionary therapy is used to address specific problems. Nevertheless, using a step-up strategy from minimally invasive treatments, we provided interventional therapy to patients with infected necrotizing pancreatitis who also had a suspected or confirmed infection and a worsened general state.

Beginning in 2011, we implemented a Continuous Regional Arterial Infusion (CRAI) using a combination of biapenem and nafamostat mesylate. Although CRAI has significantly decreased the pancreatic infection and SAP death rates, the 2016 Japanese guidelines have acknowledged that its effectiveness has not yet been proven. Therefore, we discontinued the use of CRAI in patients with SAP in 2016.

Evaluation of the psoas, abdominal muscle and fat regions

Upon enrollment, we used CE-CT to assess the pancreas (Our Hospital) and gauge the AP severity. Axial CT slices were used to assess the Visceral Fat Area (VFA) and Subcutaneous Fat Area (SFA) at the umbilical level and psoas muscle region at the level of the third lumbar vertebra (L1). VFA was colored red, whereas SFA was colored blue, according to the image analysis measured using slice Omatic software (version 5.0; Tomovision, Magog, Canada) to quantify bodily tissues.

A Hounsfield unit threshold of 150–30 was used for fat area. The psoas muscle and abdomen were manually traced to determine their size. Y. H. measured each photograph. The Psoas Muscle Index (PMI) was determined by dividing the psoas muscle area by height squared [20].

Statistics for analysis

The SPSS statistics program 26 (version 26.0; Armonk, NY, UAS) from IBM Corp. was used for the data analysis and figure production. P-values were classified as marginally significant if they were less than 0.1 and statistically significant if they were less than 0.05. To compare categorical variables—which are expressed as percentages or integers—the Chi-square test was applied. The Kolmogorov-Smirnov test was employed to confirm the normality of the continuous data distribution. When displaying continuous data, the median (Interquartile Range [IQR]) is used. We used the Mann-Whitney U-tests or the Kruskal-Wallis H-tests to compare groups where the data was not normally distributed. After statistically significant differences were discovered across the three groups, post hoc analysis was performed with the Bonferroni adjustment. The risk factors for SAP or MSAP were evaluated by applying univariate and multivariate logistic regression analyses. P-values that were less than 0.1 in univariate analysis and were statistically significant were included in multivariate analysis. For the prognostic analysis, all body parameters were split into high and low subgroups based on the median value of different tissues as the cutoff. Stepwise Cox regression analysis was utilized to identify body composition parameters highly linked with 3-month survival in individuals with SAP and MSAP. Similarly, we used Cox regression analysis to assess if the rate of body composition decrease was associated with a higher chance of recurrence one year later.

Results

Baseline features and a body composition: Examination of controls and patients with acute pancreatitis shown in Table 1.

In the sample, there were 392 patients (61.5% male; median age 54 years [IQR 40–71]) and 309 controls (62.1% male; median age 49 years [IQR 36–61]). The etiology of AP was found to be biliary in 189 patients (48.21%), hyperlipidemia in 74 patients (18.88%), other in 21 patients (5.36%), and other in 108 patients (27.55%). Acute pancreatitis associated with diabetes and hypertension was reported in 144 and 86 cases, respectively. Three hundred forty-one people had had their first episode of acute pancreatitis, whereas 51 had had previous episodes concerning body composition characteristics. Compared to controls, AP patients had a significantly higher BMI (24.98 [IQR 22.49–28.06] *vs.* 23.36 [IQR 21.46–25.75]; p<0.001).

The results showed that AP patients had significantly higher VAT (144.25 [IQR 95.10–198.88] *vs.* 97.81 [IQR 39.54–167.00]; p<0.001) and SAT (135.00 [IQR 101.53–185.20] *vs.* 120.00 [IQR 87.95–161.50]; p<0.001) when compared to controls. The study found that patients with higher amounts of adipose tissue and lower levels of attenuation (muscle and adipose tissue) had a higher risk of developing AP.

As in Table 2 The number of patients we enrolled with mild, moderately severe, and severe AP is shown in.

In Table 2: 178, 161, and 53, in that order. The median age increased along with the AP severity but did not significantly differ between the three groups. Additionally, there was no appreciable variation in the distribution of sexes. SMA (39.90 [IQR 98.67–154.15], 38.37 [IQR 30.28–44.41] and 32.25 [IQR 26.59–41.36]; p=0.003) and VAT (138.8 [IQR 92.08–183.43], 150.2 [IQR 90.22–202.25] and 166.4 [IQR 112.65–224.65]; p=0.014) showed significant differences between the three groups. By using post hoc analysis, SMA and VAT were significantly higher in the severe group as compared to the moderate group (p=0.002, p=0.013).

Table 1: Baseline features of control and patients with acute pancreatitis.

Parameters	AP (n=392)	Control (n=309)	*P*-value
Male sex, n(%)	241 (61.5)	192 (62.1)	0.859
Age (years)	54 (40–71)	49 (36–61)	<0.001
BMI (kg/m^2)[a]	24.98 (22.49–28.06)	23.36 (21.46–25.75)	<0.001
VAT (cm^2)[a]	144.25 (95.10–198.88)	97.81 (39.54–167.00)	<0.001
VATA (HU)[a]	−94.24 (−98.82~ −87.22)	−92.15 (−97.13~ −83.33)	0.002
SAT (cm^2)[a]	135.00 (101.53–185.20)	120.00 (87.95–161.50)	<0.001
SATA (HU)[a]	−99.22 (−103.1~ −93.76)	−98.01 (−101.45~ −91.92)	0.003
SMT (cm^2)[a]	123.00 (98.59–160.88)	128.30 (103.65–156.37)	0.491
SMA (HU)[a]	38.73 (30.94–44.57)	45.13 (38.62–50.07)	<0.001
L3-SMI (cm^2/m^2)[a]	43.66 (37.62–55.20)	44.50 (38.59–51.99)	0.842
Smoking, n(%)	93 (23.7)	70 (22.7)	0.739
Drinking, n(%)	98 (25)	62 (20.1)	0.122
Etiology, n(%)			
Biliary	189 (48.21)		
Hyperlipidemia	74 (18.88)		
Alcoholic	21 (5.36)		
Other	108 (27.55)		
Hypertension, n(%)	133 (33.93)		
Diabetes, n(%)	86 (21.94)		
Previous AP history	51 (13.01)		

Note: [a] Values are in median (interquartile range)

Table 2: Comparison of body composition variables according to severity of acute pancreatitis.

Variables	Severity of Acute Pancreatitis			*P*-value
	Mild (n=178)	Moderately Severe (n=161)	Severe (n=53)	
Male sex, n(%)	104(58.4)	107(66.5)	30(56.6)	0.232
Age (years)[a]	53(41–68)	54(36–71)	63(41.5–79)	0.122
BMI (kg/m^2)[a]	24.67(22.49–27.35)	25.33(22.24–28.38)	26.04(22.67–29.05)	0.249
VAT (cm^2)[a]	138.8(92.08–183.43)	150.2(90.22–202.25)	166.4(112.65–224.65)[b]	0.014
VATA (HU)[a]	−95.25(−99.23~-87.81)	−92.96(−98.24~-86.02)	−95.16(−99.39~-88.34)	0.203
SAT (cm^2)[a]	134.2(102.63–180.78)	131.5(97.02–185.60)	147.5(107.15–197.4)	0.354
SATA (HU)[a]	−99.19(−103.00~-95.25)	−98.99(−103.1~-92.82)	−99.63(−104.85~-91.54)	0.677
SMT (cm^2)[a]	119.5(98.67–154.15)	128.3(101.8–167.3)	111.5(94.73–151.85)	0.169
SMA (HU)[a]	39.90(32.78–45.71)[b]	38.37(30.28–44.41)	32.25(26.59–41.36)[b]	0.003
L3-SMI (cm^2/m^2)[a]	42.20(37.70–51.34)	45.64(38.24–56.66)	40.73(36.58–54.23)	0.106

Notes: [a] Values are in median (interquartile range). [b] Severe versus mild were statistical significance by post hoc analysis using Bonferroni adjustment

An example of a mid-L3 CT scan of an AP patient with a similar BMI is shown in Figure 1. Interestingly, VAT increased (54.01 cm^2, 135.8 cm^2, and 372.4 cm^2) in proportion to the degree of AP. Conversely, SMA (52.41 HU, 37.08 HU, and 18.99 HU) showed a tendency toward decline.

The results of the univariate and multivariate regression analyses for SAP or MSAP are shown in Table 3.

The majority of the variables did not appear to be significantly correlated with SAP or MSAP risk factors. However, the creation of SAP or MSAP was associated with SMA (OR: 0.973, 95% CI 0.953–0.993, p=0.01) and VAT (OR: 1.003, 95% CI 1.000–1.006, p=0.041). Our findings showed that for every unit increase in visceral adipose tissue, there was a 0.3% increase in risk variables for MSAP or MAP. For each unit increase in skeletal muscle attenuation, there was a 2.7% decrease in risk factors for MSAP or MAP.

Relationships between 3-month mortality in SAP and MSAP and body parameters in our population, there were no fatalities among the individuals with mild AP. We only examined the risk factors for short-term death for patients classified as SAP (dead/alive, 9/53, 17%) or MSAP (dead/alive, 2/161, 1.2%) because the literature has reported that the mortality rate for MAP patients is also relatively low [12]. For the prognosis analysis, all body parameters were entered as above and below subgroups, with the median value of different tissues acting as the cutoff. A history of recurrent acute pancreatitis, diabetes, and hypertension was also considered. There were 33 cases of recurrent pancreatitis and 181 cases of acute pancreatitis. After three months,

Table 3: Univariate and multivariate logistic regression analysis for risk factors for MSAP or SAP.

Parameters	Univariate		Multivariate	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Male sex	1.266 (0.842–1.905)	0.258		
Age	1.006 (0.995–1.016)	0.286		
BMI	1.028 (0.979–1.080)	0.272		
VAT	1.004 (1.001–1.006)	0.009	1.003 (1.000–1.006)	0.041
VATA	1.018 (0.996–1.041)	0.115		
SAT	1.001 (0.999–1.004)	0.313		
SATA	1.012 (0.989–1.036)	0.293		
SMT	1.002 (0.997–1.007)	0.501		
SMA	0.969 (0.949–0.989)	0.002	0.973 (0.953–0.993)	0.01
L3-SMI	1.008 (0.990–1.025)	0.396		
Smoking	0.740 (0.461–1.188)	0.213		
Drinking	0.697 (0.437–1.111)	0.129		
Hypertension	0.855 (0.561–1.304)	0.467		
Diabetes	0.736 (0.452–1.197)	0.217		
Previous AP history	0.617 (0.334–1.138)	0.122		

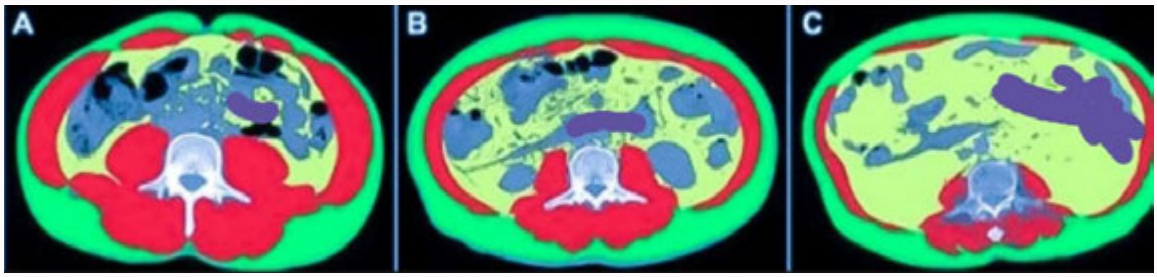

Figure 1: Computed tomographic scans with varying degrees of severity that display body composition in several AP patients. [A-C] Display AP that is mild, moderately severe, and severe, in that order. Dark red represents skeletal muscle, faint yellow represents visceral adipose tissue, and shinning green represents subcutaneous adipose tissue. When the degree of AP grew at a comparable BMI, VAT likewise steadily increased (54.01 cm^2, 135.8 cm^2, and 372.4 cm^2). Conversely, SMA displayed a trend toward decline (52.41 HU, 37.08 HU, and 18.99 HU).

the death rate for patients with sound skeletal muscle attenuation was 0.9% (dead/alive, 1/107), while the death rate for patients with low skeletal muscle attenuation was 9.3% (dead/live, 10/107). The stepwise Cox regression model revealed a significant correlation between minimal skeletal muscle attenuation and the 3-month survival risk (Hazard Ratio [HR]: 10.500, 95% Confidence Interval [CI]: 1.344–82.025, p=0.025).

The mortality rate for patients with low skeletal muscle attenuation was 9.3% (dead/alive, 10/107), whereas the mortality rate for patients with high skeletal muscle attenuation was 0.9% (dead/live, 1/107). A Cox regression analysis of body composition found that the only risk factor for 3-month death was poor skeletal muscle attenuation (Hazard Ratio [HR]: 10.500, 95% Confidence Interval [CI]: 1.344–82.025, p=0.025).

View the entire size AP and body composition parameter relationships about 1-year recurrence in this group, 79 individuals underwent CT evaluations; 33 experienced a recurrence, and 46 did not.

Regarding Table 4 shows the patients' baseline CT scan and one-year follow-up CT scan. In the nonrecurrent group, the median VAT decreased from 144.20 (IQR 101.35–209.80) cm^2 to 103 (IQR 69.68–162.38) cm^2 (p=0.005), a statistically significant decrease. However, there was no statistically significant difference between the median VATs of the first AP and recurrent AP groups (156.6 [IQR 119.70–198.85] *vs.* 152 [IQR 100.3–186.7], p=0.442). Similarly, SAT decreased from 129.60 (IQR 95.06–192.78) cm^2 to 114.55 (IQR 72.07–170.40) cm^2 (p=0.055), a marginally significant decrease.

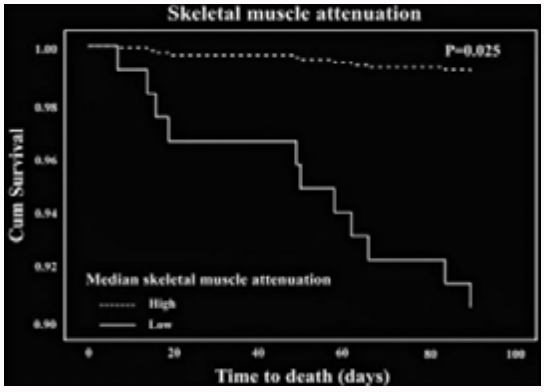

Figure 2: Relationships between AP and body composition parameters with regard to 1-year recurrence.

Table 4: Body composition features of recurrent and no-recurrent with AP by different states in 1 year.

	No-Recurrent (n=46)		P-value	Recurrent(n=33)		P-value
	Initial (AP)	Initial (Normal)		Initial (AP)	Initial (RAP)	
VAT	144.20 (101.35-209.80)	103.00 (69.68-162.38)	0.005	156.60 (119.70-198.85)	152.00 (100.30-186.70)	0.442
SAT	129.60 (95.06-192.78)	114.55 (72.07-170.40)	0.055	152.00 (110.70-182.25)	148.30 (97.47-185.65	0.577
SMT	118.90 (97.15-145.80)	106.05 (93.38-141.98)	0.41	126.80 (95.49-162.30)	129.70 (91.02-157.10)	0.858
SMA	37.42 (28.69-43.30)	35.74 (29.92-43.90)	0.821	37.67 (31.56-44.31)	40.28 (30.84-44.69)	0.677
L3-SMI	41.20 (37.43-50.15)	39.41 (36.33-48.65)	0.29	42.86 (37.25-57.07)	43.16 (35.46-56.03)	0.893

Table 5: Association between rate of body composition loss within 1 year recurrent, according to univariate cox regression analysis.

Parameters	Hazard Ratio	95% Confidence Interval	*P*-value
Male sex	0.11	0.511–2.413	0.791
Age	0.995	0.977–1.013	0.572
VAT rate of loss (>median)	0.427	0.189–0.967	0.041
SAT rate of loss (>median)	0.604	0.295–1.238	0.168
SMT rate of loss (>median)	0.887	0.429–1.832	0.745
SMA rate of loss (>median)	0.874	0.436–1.754	0.705
L3-SMI rate of loss (>median)	0.887	0.429–1.832	0.745

The rate of body composition loss (%) was computed using the ratio of the difference value between the baseline and follow-up CT scans and the beginning body composition value. Patients with visceral adipose tissue loss greater than 17% had a recurrence rate of 23% (9/39), while patients with less than 17% had a recurrence incidence of 60% (24/40). The VAT decrease's median percentage was 17%. To ascertain the risk ratios connected to the emergence of RAP, Cox regression analysis was employed. The results showed that visceral adipose tissue loss greater than 17% prevented a 1-year recurrence (Hazard Ratio [HR] 0.427; 95% CI 0.189–0.967, P=0.041) showed in (Table 5).

Discussion

Body composition parameter analysis is a relatively new approach that can be easily obtained from Computed Tomography (CT) images. CT has become increasingly important in the diagnosis, treatment, and follow-up of patients with AP. Our research revealed a significant relationship between the VAT and SMA as determined by CT and the advancement of SAP or MSAP. There was a correlation between SMA and the death rate in patients with SAP and MSAP. Notably, visceral adipose tissue loss of at least 17% within a year was protective against recurrent AP. Understanding body composition may help with clinical outcome prediction. Many AP studies have not included control groups. This study found that AP patients had less adipose tissue attenuation than controls [15]. Points out lower adipose tissue attenuation is linked to higher lipid content and larger adipocytes. This result was consistent with the observation that, in comparison to the control group, AP patients exhibited greater levels of visceral (144.25 cm^2) and subcutaneous (135 cm^2) adipose tissue. Like the control group, the AP patients' SMA was lower (38.73 *vs.* 45.13 HU). It has been shown that low SMA, indicating myosteatosis, is a crucial component influencing muscle function [16]. Lower C, T attenuation of muscle is usually due to increased fatty infiltration of muscle, or "myosteatosis." These results suggested that high adipose tissue and low attenuation (muscle and adipose tissue) could be risk factors for developing AP. This study indicates that SMA was crucial to creating SAP, or MSAP. For every unit improvement in skeletal muscle attenuation, there was a 3.1% decrease in risk variables for MSAP or MAP. The one study we could locate [16], suggested that low mean muscle attenuation was a risk factor for SAP. Still, it did not elaborate on how this relationship altered the degree of AP. We proposed the following theories as possibilities: Leptin's primary action on the hypothalamus suppresses hunger and restricts the amount of fat accumulating in peripheral tissues, such as skeletal muscle [17-19,21]. High leptin levels would keep skeletal muscle attenuation high by preventing lipid buildup in the muscle. High levels of skeletal muscle attenuation and high amounts of leptin may be positively correlated, according to recent research. Leptin also possesses anti-inflammatory qualities [22]. Skeletal muscular function is reduced in patients with low SMA. In addition, skeletal muscle produces less leptin, which lessens its anti-inflammatory properties [22]. We also examined mortality risk variables for patients with SAP and MSAP. Exogenous leptin supplementation has decreased NO expression, proinflammatory factor expression, and inflammation levels in animal tests, all of which lessen the severity of pancreatitis. This patient group's death rate (11/214) was 5.1%, similar to the 5.4% reported rate [23]. Studies indicate a high correlation between death from esophageal cancer, extremity sarcomas, cirrhosis, and poor SMA, as determined by CT [24-26]. In addition, investigations have indicated that patients with pancreatic cancer with poor skeletal muscle attenuation—as opposed to low muscle mass—have a reduced prognosis [27]. These results are consistent with earlier studies. Low SMA most likely results in an imbalance in adipokines and other cytokines, which can exacerbate inflammation, impair immunity, and increase mortality risk [28]. VAT had a big impact on how SAP or MSAP patients developed. High levels of adipose tissue deposition were closely associated with a chronic inflammatory state. Leptin function can be hampered, and leptin resistance can ensue from chronic inflammation's disruption of leptin receptor signaling. According to [29], the hypothalamus's leptin resistance can make it difficult to regulate weight, which may result in obesity. Furthermore, leptin resistance can lessen how leptin inhibits the buildup of lipids in skeletal muscle, which may cause patients to develop myosteatosis or sarcopenia. Studies have shown that patients with high VAT

who had pancreatic cancer had an increased risk of surgery site infections [30]. Studies have also shown a negative correlation between abdominal obesity and serum adiponectin levels [31]. The anti-inflammatory effects of serum adiponectin are well established [32]. A vicious cycle occurs when IL-6 and TNF-α prevent adipocytes from producing adiponectin [33]. Serum adiponectin also increases mitochondrial fatty acid oxidation and free fatty acid elimination [34]. Low adiponectin levels cause lipotoxicity by reducing the removal and lipid oxidation of fatty acids. Adiponectin stimulates immune cells to produce anti-inflammatory mediators and prevents macrophages from producing proinflammatory cytokines (TNF-α and IL-6) [31]. Appropriate animal experimental research has shown that adiponectin treatment can considerably lessen the severity of AP [35]. In the recurrent AP trial, Table 4 shows that VAT remained high in the recurrent group (156.6 *vs.* 152 cm^2, p=0.442) and showed little variation from the initial measurement [31]. Patients with SAP have been found to have much lower adiponectin levels than patients with MAP. In contrast, the VAT of the nonrecurrent group dropped significantly (p=0.005), from 103 cm^2 to 144.2 cm^2. A recent study found that during follow-up, there is a positive link between abdominal obesity in AP patients and higher levels of two proinflammatory cytokines, TNF-α and IL-6 [36]. Our findings are consistent with this one. Furthermore, the statistics suggested that a decrease in VAT of over 17% in less than a year acted as a buffer. Important variables in this study include treatment regimens and post-discharge health education for AP patients about exercise and diet. The main therapeutic options for people with AP are enzyme inhibition, acid inhibition, gastrointestinal decompression, fasting water, and other physiological requirements. For patients with MSAP and SAP, it is essential to block SIRS, preserve organ function, provide analgesia, initiate enteral feeding early, use antibiotics responsibly, manage local and systemic problems appropriately, and undergo surgical treatment [37]. Out of the individuals in our dataset, 14 had surgery. Finally, the doctor advised the patients to maintain a nutritious diet and moderate exercise after being released from the hospital. It has been proposed that consuming vegetables, salmon, fiber, and milk can reduce the incidence of AP [38-40]. Physical activity and the incidence of AP were found to be inversely correlated in prospective cohort research with 0.5 million individuals [41]. Furthermore, it has been proposed that combining resistance and aerobic training can enhance muscular function and reduce the prevalence of obesity-related sarcopenia [42]. To avert the illness and enhance their chances of recovery, patients must adhere to a sensible diet and exercise schedule. We acknowledge that there are several restrictions on our study. At first, the study used a retrospective approach and failed to find any cytokines that were either pro- or anti-inflammatory. Second, once they recover, many patients do not have their abdomen CT scans reexamined, which may lead to errors when conducting follow-up tests. Third, the CT images were measured by a lone, knowledgeable physician [10,13]. Lastly, only a single-center population in China was included in this study. Our measurement has minimal bearing on the outcomes because it has been observed that the correlation coefficient between the two assessments may reach 0.954 to 0.99. Further multicenter, large-sample randomized prospective studies in different settings will be helpful to validate our findings.

Conclusion

Attenuation of visceral adipose tissue and skeletal muscle was highly correlated with the severity of AP and the prognosis of patients. To find out how VAT and skeletal muscle attenuation impact the course and outcome of AP, more research is needed. Finally, patients should be educated on good food and activity after discharge. Patients can enhance their quality of life and prognosis by improving their muscle function and losing visceral fat with a good diet and exercise regimen.

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