

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

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1. Abstracts

1.1. Background: The association between abdominal visceral fatty area (VFA) and muscle mass and mortality is not fully understood despite the fact that being overweight is an established risk factor for the onset and severity of acute pancreatitis (AP). We assessed the effect of VFA on severe AP (SAP) mortality

The aim of this study was to investigate the association of adipose and muscle parameters with the severity grade of AP

1.2. Methods: Between April 2011 and March 2021, 238 consecutive patients with SAP were assessed in this retrospective, our facility cohort study.

We enrolled 454 patient subgroups for the first contrast-enhanced computed tomography for muscle tissue parameters in addition to adipose tissues and concluded that severity was analyzed through logistic regression analysis. The predictive capacity of the parameters was investigated using receiver operating characteristic (ROC) curves.

Computed tomography (CT) was used to evaluate VFA umbilical level L1. And the effects of visceral adiposity and muscle parameters on AP mortality.

1.3. Results: Our Egyptian participants, 66% were men and the median age was 126. Eighteen patients (7.5%) died during hospitalization. Twelve obese individuals had a body mass index (BMI) >30 kg/m² (5%) with a median BMI of 22.2 kg/m². The median VFA was 112 cm², and the waist measurement was 85.5 cm. A total of 176 (57.1%) individuals had VFA larger than 100 cm².

VFA (COV, 167 cm²; AUC = 0.679), accompanied by a prognostic factor score based on the Japanese guidelines for AP care (cutoff value [COV], 4; area under the curve [AUC] = 0.869) and age [COV, 72; AUC = 0.780]), demonstrated considerable accuracy in predicting death. Elevated VFA was associated with a significantly higher odds ratio (OR) for predicting mortality according to univariate regression analysis, but not multivariate analysis (OR:4.38, P = 0.0406). There was no discernible difference in the survival periods of patients with SAP with and without an elevated VFA of 167 cm².

Regarding to employed subgroup's for assess the muscle mass effect we found no distinct variation was found between the AP severity groups in either adipose tissue parameters (visceral adipose tissue and subcutaneous adipose tissue) or visceral muscle ratio. However, muscle mass and mean muscle attenuation differed significantly, with p-values of 0.037 and 0.003, respectively. In multivariate analysis, low muscle attenuation was associated with severe AP with an odds ratio of 4.09 (95% confidence intervals:1.61–10.36, p-value 0.003). None of the body parameters showed sufficient predictive capability in the ROC curve analysis.

1.4. Conclusions: Visceral obesity did not significantly affect the ability to predict mortality in SAP patients. Low muscle attenuation is associated with an increased risk of developing severe AP. Future prospective studies will help identify the underlying mechanisms and characterize the influence of body composition parameters on AP.

1.5. 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 health-care systems. Research on the relationship between obesity and acute pancreatitis severity has produced inconsistent findings.

1.6. 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.

2. 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 15–25% of AP patients progress to significant SAP, 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 [11,12-19].

In brief, the outcomes of earlier research exhibit heterogeneity, and there are insufficient data from sufficient studies to draw broad generalizations for specific parameters.

In this extensive multicenter European study, we aimed to assess the role of muscle and adipose factors in the progression of AP severity.

3. Material and Methods

3.1. Researchers and Methodology

This study analyzed consecutive patients with SAP at the Zagazig University Hospital from April 2011 to March 2021 as part of a retrospective, single-center cohort. In accordance with the Japanese guidelines for the management of AP, AP was diagnosed based on an environment with any two of the following three criteria: acute pain in the upper abdomen, an increase in serum amylase >3 times or lipase, and characteristic features of AP upon imaging [9]. As soon as an AP determination was made, we repeated severity evaluations within 24 h and after 24-48 hours using the

seriousness score system recommended by the Japanese Ministry of Health, Labour, and Welfare [9]. The Japanese severity criteria for AP are, in brief, comprised of two factors: (i) prognostic factors with a total point score based on nine physiological and laboratory measurements, including age, platelet count, serum calcium, C-reactive protein (CRP), blood urea nitrogen (BUN), lactate dehydrogenase (LDH), and bass excess; and (ii) computed tomography (CT) grade determined by contrast-enhanced CT (CE-CT) [9]. When the combined prognostic factor score was ≥ 3 , the CT grade was ≥ 2 , or an assortment of these factors was present, SAP was identified [9]. In the current study, we included patients with SAP, but we excluded those who had been diagnosed at a different hospital before being transferred or those who had not undergone a CE-CT evaluation at the time of enrollment.

Characteristics of body composition:

All patients had their first CECT performed at the start of AP. OsiriX Lite v9.0 (Pixmeo SARL, Geneva, Switzerland) and the DCM tool were used to quantify the parameters of adipose tissue and muscle, for which excellent inter-observer agreement has previously been reported. Following the selection of a cross-sectional slice at the level of the L3 vertebral body, regions of interest (ROI) containing the muscle and adipose tissue compartments were manually defined (Figure 1). Measurements in this region were not disregarded because visible peripancreatic necrosis and stranding are infrequently observed. The ranges of -190 to -30 Hounsfield units (HU) for adipose tissue and -30 to 150 for muscle tissue were used to select tissues within the ROIs, as previously reported. Refer to Supplementary Figures A–E for depicting individual ROIs. A = MM/VAT is VMR. From the histogram, which shows an exemplary histogram in Supplementary Figure 1, A–E, the mean muscle attenuation (MMA) was retrieved in the range of -29 to $+150$ HU. Only CT scans conducted in the native or venous phase were used in this study, because the CT phase affects the measurement of myosteatosis. In addition to a radiologist with expertise in gastroenterological radiology (NL), two independent investigators (MJM and LEK) performed all analyses. 35 CECTs, or 7.7% of the entire cohort) were randomly selected and blindly reanalyzed using the MJM to assess the inter-observer variability of the analyses. All parameters had interobserver agreement coefficients between 0.873 and 1.0, with a mean value of 0.954.

As shown in Figure 1, the DCMTool was used to visualize and define the regions of interest (ROI) of the various bodily compartments. The compartments used to measure fat content are shown in (a). The space between the blue lines represents the subcutaneous compartments. The green line indicates the location of the visceral compartment. (b) Muscle sections. The back muscles are shown in green, the psoas muscle in red, and the abdominal muscles in blue. The supplementary files can be viewed for further details of the measurements.

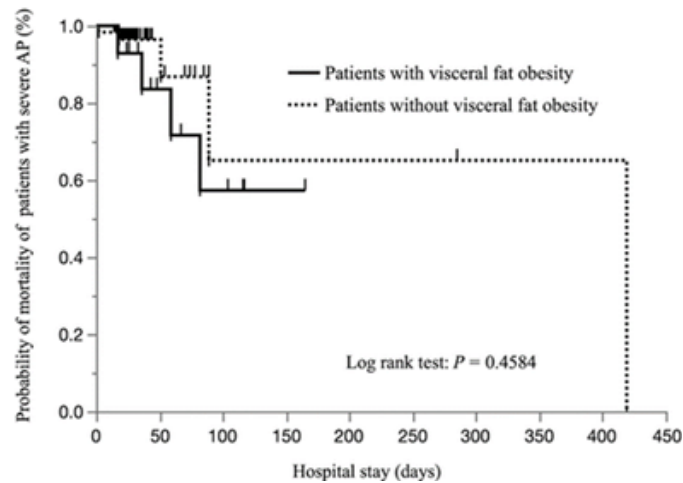


Figure 1: shows the general survival curves of patients with severe pancreatitis (AP) who had higher VFA than those who did not.

3.2. 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 hours 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.

3.3. 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 software SYNAPSE VINCENT (Fujifilm, computerized, Zagazig Technology). 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 [10].

3.4. Statistical Evaluation

For continuous variables, data are shown as median and interquartile range (IQR), and categorical variables are presented as absolute numbers and percentages. The Mann-Whitney U test was used for continuous data, and the chi-square test was used for categorical variables. Since normalcy was not assumed, group comparisons were made using Mann-Whitney U or Kruskal-Wallis tests. Univariate and multivariate logistic regression analyses were used to examine potential relationships between the measured parameters and severe AP. The data were subjected to multivariate analysis to account for sex and age. Age was dichotomized into an upper interquartile range (73 years). In addition, the body composition parameters were divided into three groups (tertiles) in both univariate and multivariate analyses according to the lowest, medium, and highest IQR levels of each parameter. Regression analysis results are displayed as odds ratios (ORs) and adjusted odds ratios (aORs), together with the appropriate p-values and 95% confidence intervals (CIs). Receiver operating characteristic (ROC) curves and related areas under the curve were used to assess the predictive power of the measured parameters. A statistically significant p-value of ≤ 0.05 was considered in all analyses.

Version 24 of the SPSS statistics program (IBM Corp., Armonk, NY, USA) was used for all computations.

4. Results

4.1. Basic Traits and Subsequent Trajectory

We admitted 888 patients with AP to our hospital, of whom 454 had studied the effect of muscle mass on the severity of pancreatitis.

A single patient did not undergo CE-CT upon admission, although 240 patients (27%) had severe disease. A total of 238 patients were included in this study. Table 1 shows participants' initial demographic characteristics. The participants were 66% male and had a median age of 62 years (IQR:50–74 years). Cholelithiasis was the cause of AP in 100 (42%) patients. Alcoholics was the etiology in 74 (31%) patients, idiopathic disease in 22 (9%), and other factors.

Twelve obese individuals (5% of the total) had a BMI > 30 kg/m², with a median BMI of 22.2 kg/m². Median waist size was 85.5 cm, VFA was 112 cm², and SFA was 114 cm², respectively. Indicative of the danger of obesity-related illnesses in Egyptian people as a diagnostic criterion for being overweight, there were 136 (57%) individuals with a VFA exceeding 100 cm².¹¹ The PMI was 4.7 kg/m², and the psoas muscle area at the third lumbar vertebra (L3) level was 12.5 cm².

Table 2 presents the participants' severity scores. The median prognostic factor score and CT severity score were one and two following the Japanese recommendations. ICU stay and hospitalization were, on average, 4 days and 29 days, respectively. A total of 66 patients (28%) experienced local problems during their hospital stay, including four with APFC, two with ANC, 24 with PPC, and 32 with WON. Eighteen patients (7.5%) died during hospitalization.

Risk factors for persons with SAP who will die

Using the ROC curve analysis, we assessed the COVs of various clinical and physical characteristics to predict mortality. Our calculations yielded COVs of 4, 72 years, 20.5 kg/m², 88.8 cm, 167 cm², 143 cm², 7.2 cm², and 5.32 kg/m² for the prognostic factor score, age, BMI, waist circumference, VFA, SFA, and psoas muscle area. VFA (AUC = 0.679) and the prognostic factor score had the highest accuracy in predicting death in patients with SAP, whereas BMI, waist circumference, SFA, psoas muscle area, and PMI had the lowest accuracy (Table 3). The prognostic factor score and age had a moderate accuracy > 0.7 in the area under the curve.

According to a univariate logistic analysis employing medical parameters, greater predictive factor scores (OR:20.0, 95% CI, 4.33-92.4, P = 0.00001), older age (OR:22.3, 95% CI, 2.68-186, P = 0.0041), and larger VFA (OR:4.38, 95% CI, 1.67-18.0, P = 0.0406) were important for predicting death rates. Three important elements were used in the multivariate evaluation. Age and prognostic factor scores were substantial for themselves (Table 4) but not for VFA (P = 0.4258). Figure 1 displays a survival rate map of 119 patients with SAP, with and without visceral obesity, and with a VFA > 167 cm². There was little difference between the two groups.

We also examined whether these important risk factors were associated with shorter hospitalizations (≥ 30 days) and ICU stays (≥ 10 days) in patients with SAP. The predictive factor score and greater VFA were significant for ICU stays < 10 days, whereas

older age was beneficial for hospitalizations ≥ 30 days according to univariate regression analysis. The predictive factor score was found to be an important risk factor for ICU stay ≥ 10 days by multivariate logistic regression analysis, whereas VFA was only marginally significant (P = 0.0707) (Table 5).

4.2. Local Problems, Bodily Parameters, and Clinical Data

Of the 66 patients with localized morbidity, 26 underwent suppuration and 40 did not undergo suppuration. Moreover, elderly patients with comorbidities had suppuration (P = 0.0645); however, there were no related factors among the predictive factor score, age, VFA, and local complications (Table 6).

4.3. Outcomes of Subgroup

A total of 454 patients (58.1%) were male. Table 7 summarizes the baseline physical characteristics of each patient according to disease severity. Most of the patients had modestly severe illnesses (49.6%). Age and BMI disparities in the severity classes were not statistically significant. The most frequent etiology (41.6%) was alcohol-induced pancreatitis brought on by alcoholism. Of the 30 patients, 6.6% had an overall death rate, with 25.1% developing transient or permanent organ failure.

Features of patient, Table 7.

The impact of 454 patients' muscle mass and adipose tissue on the severity of their pancreatitis was investigated using CT analysis. The summ of every patient under study served as the inclusion and exclusion criterion.

completed moderately severe [N] = 454, N = 163 at 35.9%, 225 at 49.6%, and 66 (14.5%) at %) Women's (584.8) Men 15.2 26436.8 132 (50.0) 92 (34.8) 41.90 (40190) 37.3 out of 71 female members 93, 4 (26 13.7) 48.9 sixty (46-73) sixty.0-(49-74) -56 (46-68) years (48-77) -63 kilos per square meter (BMI) 24.7-81.1) 26.7 31.0 - 24.8 = 27.7 Two 28.8 (27.5; 24.3-30.8) = 4.8-31.1 #%#) The cause Endocrine 41.6, or 189 44.7% of 74 (45.4) = 96 (19 (28.1) Beverages 13.5 (29.7) Forty (24.5) 22 (33.3) and 73 (32.4) Sneaky 26 (22.1) 19(6), 4413.7 (19.7), 99 (30.5) Those Others 12 (5.3), 38 (8.1) 13 = 7.0182.2(%) 442.1) 66 (100) 114 (25.1) Failed Organs [30 (6.6)] of fatalities, or 0% * 0.6 * 1.8 = 1.31 (37.9) * 3 (1-7) * When CT will occur (in days) 3-5; 4-9; 1-3; 1-7 *The numbers represent the median (IQR).

Etiology breakdown according to severity categories

The BMI, or body mass index, is expressed as kg/m².

Table 1: Patients with severe acute pancreatitis have certain traits.

	N = 119
Age (IQR), years	62 (50–74)
Male sex, n (%)	158 (66.4)
Etiology	
Cholelithiasis, n (%)	100 (42.0)
Alcoholic, n (%)	74 (31.1)
Pancreatolith, n (%)	12 (5.0)
Post-ERCP, n (%)	8 (3.4)
Drugs, n (%)	6 (2.5)
Hyperlipidemia, n (%)	6 (2.5)
Idiopathic disease, n (%)	22 (9.2)
Others, n (%)	10 (4.2)
Body parameters	
Body weight (IQR), kg	60 (52–70)
Height (IQR), cm	165 (154.2–171)
BMI (IQR), kg/m ²	22.2 (20.6–24.6)
BMI ≥30 kg/m ² , n (%)	12 (5.0)
Waist circumference (IQR), cm	85.5 (79.8–91.8)
VFA at the umbilical level (IQR), cm ²	112 (65.7–165)
VFA at the umbilical level ≥100 cm ² , n (%)	76 (57.1)
SFA at the umbilical level (IQR), cm ²	114 (81.2–159)
VFA at L3 level (IQR), cm ²	110 (57, 180)
VFA at L3 level ≥100 cm ² , n (%)	130 (54.6)
SFA at L3 level (IQR), cm ²	92 (64, 130)
Psoas muscle area, cm ²	12.5 (9.54–16.3)
Psoas muscle index (IQR), cm ² /m ²	4.7 (3.8–5.5)

Table 2: Patients with profound acute pancreatitis and their clinical outcomes

Outcome Severity outcomes	N = 119
Prognostic factor score, median (IQR)	1 (0.25, 3)
0, n (%)	62 (26.1)
1, n (%)	74 (31.1)
2, n (%)	38 (16.0)
3, n (%)	32 (13.4)
4 or more, n (%)	32 (13.4)
CE-CT grade	
Grade 1, n (%)	20(8.4)
Grade 2, n (%)	196 (82.4)
Grade 3, n (%)	22 (9.2)
Clinical outcomes, median (IQR)	4 (2, 2)
ICU stay (IQR) days	8 (2, 8)
ICU stay ≥10 days, n (%)	44 (18.5)
Hospital stay (IQR) days	58 (20, 44)
Hospital stay ≥30 days, n (%)	86 (36.1)
Hospital mortality, n (%)	18 (7.6)
Treatment	
Enteral nutrition, n (%)	158 (66.3)
Continuous regional arterial infusion, n (%)	46 (19.3)
EUS-CD, n (%)	10 (4.6)
Percutaneous drainage, n (%)	10 (4.6)
Continuous hemodiafiltration, n (%)	4(1.8)
Plasmapheresis, n (%)	2 (0.9)
Complication	
Local complication, n (%)	66 (27.7)
Infected/sterile, n (%)	26 (10.9)/40 (18.5)
Acute peripancreatic fluid collection, n (%)	8 (1.7)
Acute necrotic collection, n (%)	2 (0.84)
Pancreatic pseudocyst, n (%)	24 (11.8)
Walled-off necrosis, n (%)	32 (13.4)
Thrombogenic events, n (%)	20 (18.2)

CE-CT, contrast-enhanced computed tomography; EUS-CD, endoscopic ultrasonography-guided pancreatic cyst drainage; ICU, intensive care unit; IQR, interquartile range.

Table 3: Lists the cutoff values, sensitivity, and specificity of clinical indicators that indicate death for people with severe acute pancreatitis.

	COV	AUC	Sensitivity	Specificity
Prognostic factor score	4	0.869	0.667	0.909
Age, years	72	0.78	0.889	0.736
BMI, kg/m ²	20.5	0.537	1	0.246
Waist circumference, cm	88.8	0.538	0.778	0.64
VFA at the umbilical level, cm ²	167	0.679	0.556	0.736
SFA at the umbilical level, cm ²	143	0.521	1	0.346
VFA at the L3 level, cm ²	158	0.669	0.667	0.709
SFA at the L3 level, cm ²	105	0.561	0.889	0.373
Psoas muscle area, cm ²	7.2	0.564	1	0.291
Psoas muscle index	5.32	0.548	1	0.3

AUC, area under the curve; BMI, body mass index; COV, cutoff value; PMA, psoas muscle area; SFA, subcutaneous fat area; VFA, visceral fat area.

Table 4: Univariate and multivariate logistic regression (LR) study of risk factors for deaths for individuals with serious, acute pancreatitis.

	Univariate Odds ratio (95% CI)	P-value	Multivariate Odds ratio (95% CI)	P-value
Prognostic factor score ≥ 4	20.0 (4.33–92.4)	0.00001	8.57 (1.57–46.6)	0.013
Age ≥ 72 years	22.3 (2.68–186)	0.0041	12.5 (1.37–114)	0.0251
VFA at the umbilical level ≥ 167 cm ²	4.38 (1.67–18.0)	0.0406	2.04 (0.353–11.8)	0.4258

VFA, visceral fat area

Table 5: shows the univariate and multivariate logistic regression analyses of the risk factors for hospitalization for less than 30 days in patients with severe acute pancreatitis.

	Univariate Odds ratio (95% CI)	P-value	Multivariate Odds ratio (95% CI)	P-value
ICU stay ≥ 10 days				
Prognostic factor score ≥ 4	4.56 (1.47–14.1)	0.0084	3.46 (1.05–11.4)	0.0418
Age ≥ 72 years	1.34 (0.507–3.54)	0.5549		
VFA ≥ 167 cm ²	3.69 (1.30–10.5)	0.0145	2.79 (0.917–8.46)	0.0707
Hospital stay ≥ 30 days				
Prognostic factor score ≥ 4	2.61 (0.895–7.60)	0.0789		
Age ≥ 72 years	3.02 (1.35–6.76)	0.0073		
VFA ≥ 167 cm ²	2.27 (0.873–5.89)	0.0926		

ICU, intensive care unit; VFA, visceral fat area

Table 6: provides information on local complications based on age, VFA, and the Japanese Severity Score (JSS).

Local complication	Total	Total	With Infected	Sterile	P-value [†]	Without	P-value [‡]
Prognostic factor score							
≥ 4	16	10	5	5	0.4611	6	0.0019
< 3	103	23	8	15		80	
Age (years)							
≥ 72	37	11	7	4	0.0645	26	0.8257
< 72	82	22	6	16		60	
VFA (cm ²)							
≥ 167	21	6	3	3	0.6588	15	1
< 167	98	27	10	17		71	

[†] Compared with infected and sterile local complications.

[‡] Compared to patients with and without local complications.

Table 7: Patient severity characteristics.

	Total N=454	Mild N=163 (35.9%)	Moderately severe N=225 (49.6%)	Severe N=66 (14.5%)
Gender (%)				
Male	264 (58.1)	92 (34.8)	132 (50.0)	40 (15.2)
Female	190 (41.9)	71 (37.4)	93 (48.9)	26 (13.7)
Age (years)*	60 (46–73)	63.0 (49–74)	56 (43–68)	63 (48–77)
BMI (kg/m2)*	27.7 (24.8–31.1)	27.7 (24.8–31.0)	27.8 (24.8–31.1)	27.5 (24.3–30.8)
Aetiology (%#)				
Biliary	189 (41.6)	74 (45.4)	96 (42.7)	19 (28.1)
Alcohol	135 (29.7)	40 (24.5)	73 (32.4)	22 (33.3)
Idiopathic	93 (20.5)	36 (22.1)	44 (19.6)	13 (19.7)
Others	37 (8.1)	13 (7.0)	12 (5.3)	12 (18.2)
Organ failure (%)	114 (25.1)	0	48 (42.1)	66 (100)
Mortality (%)	30 (6.6)	1 (0.6)	4 (1.8)	25 (37.9)
Timing of CT (days)*	3 (1–7)	3 (1–5)	4 (1–9)	3 (1–7)

Table 8: Relationship between acute pancreatitis severity and body composition metrics.

	All	Mild	Moderately severe	Severe	p-value
VAT					
Median	217.3	221.2	208.1	238.8	0.10a
IQR	139.7–290.5	144.9–283.6	131.7–287.5	172.1–333.5	
SAT					
Median	209.2	204.5	213.5	212	0.97a
IQR	145.0–285.1	151.9–274.8	141.6–289.6	144.8–281.6	
MM					
Median	129.5	126.6	130	136.9	0.037a
IQR	105.3–158.6	100.1–153.4	108.0–160.1	108.1–169.4	
MMA					
Median	29.3	29.8	29.6	23.4	0.003a
IQR	22.2–36.6	22.4–37.8	23.2–37.1	18.3–32.1	
VMR					
Median	1.64	1.76	1.57	1.73	0.14a
IQR	1.14–2.18	1.14–2.23	1.04–2.11	1.27–2.38	

Kruskal-Wallis test notes: VAT, SAT, MM, and VMR in cm²; MMA in HU; MM and MMA indicate muscle attenuation.

Table 9: Univariate and multivariate regression analysis of risk factors for severe acute pancreatitis. Age was dichotomised at 73 years (highest tertile in the IQR).

Logistic regression analysis of risk factors for severe AP				
	Univariate OR (95% CI)	p-value	Multivariate aOR (95% CI)	p-value
Gender				
Female	1 (ref)			
Male	1.126 (0.661–1.920)	0.662	1.217 (0.682–2.172)	0.505
Age				
<73 years	1 (ref)			
≥73 years	1.609 (0.916–2.824)	0.098	1.495 (0.815–2.745)	0.194
BMI				
>25.0	1 (ref)			

18.5–24.9	0.998 (0.517–1.863)	0.528	0.981 (0.517–1.863)	0.954
<18.5	0.001 (0.000–0.002)	0.001	0.001 (0.000–0.002)	0.001
VAT				
Highest tertile	2.242 (1.031–4.876)	0.042	2.157 (0.953–4.883)	0.065
Mid tertile	1.569 (0.761–3.234)	0.222	1.551 (0.750–3.207)	0.236
Lowest tertile	1 (ref)			
SAT				
Highest tertile	0.923 (0.420–2.027)	0.841	0.921 (0.395–2.148)	0.848
Mid tertile	0.994 (.508–1.946)	0.987	0.982 (0.499–1.932)	0.958
Lowest tertile	1 (ref)			
MM				
Highest tertile	1 (ref)			
Mid tertile	0.596 (0.324–1.094)	0.095	0.496 (0.256–0.961)	0.038
Lowest tertile	0.625 (0.306–1.094)	0.199	0.432 (0.166–1.124)	0.085
MMA				
Highest tertile	1 (ref)			
Mid tertile	1.536 (0.670–3.519)	0.311	1.577 (0.683–3.640)	0.286
Lowest tertile	3.446 (1.468–8.086)	0.0004	4.085 (1.609–10.37)	0.003
VMR				
Highest tertile	1 (ref)			
Mid tertile	0.921 (0.494–1.714)	0.794	1.025 (0.538–1.954)	0.94
Lowest tertile	0.587 (0.268–1.287)	0.184	0.673 (0.300–1.508)	0.336

Abbreviations: OR, odds ratio; VAT, visceral adipose tissue; SAT, subcutaneous adipose tissue; MM, muscle mass; MMA, mean muscle attenuation; VMR, visceral muscle ratio.

Multivariate analysis: results adjusted for age and gender.

4.4. The Properties of Fat and Muscle

Of the 454 CT images examined, 183 were in the native phase, 243 were in the venous phase, and 28 were in the arterial phase. The two adipose tissue metrics, VAT and SAT, and two of the three muscle tissue metrics, MM and VMR, did not exhibit any discernible variation across sessions. As indicated in the Methods section, only native and venous CTs were used in MMA analysis. The median interval between admission and CT scan was 3 days (IQR 1-6), with no appreciable difference between the severity groups (Figure 1 and 2, supplementary file).

Table 8 presents the median values and IQRs of the measured variables.

The groups with mild, moderately severe, or severe AP did not differ significantly in terms of the VAT, SAT, or VMR. Based on p-values of 0.037 and 0.003, respectively, the differences were significant for MM and MMA. Of the men, the following were the gender-specific median values: VAT:252.6 (IQR 173.0–335.3) cm², SAT:181.5 (IQR 132.7–235.9) cm², MM:152.2 (IQR 132.0–170.3) cm², MMA:32.2 (IQR 24.4–38.6) HU, and VMR:1.64 (IQR 1.20–2.18) cm². Values that corresponded to women were as follows: VAT: 171.1 (IQR 111.7–232.5) cm², SAT: 260.1 (IQR 183.1–345.2) cm², MM: 104.6 (IQR 92.7–116.7) cm², MMA:

25.4 (IQR 19.5–32.9) HU, and VMR: 1.65 (IQR 1.05–2.18) cm². For all metrics except VMR, where there was no difference, the gender variances differed considerably (p<0.0001).

Relationship between acute pancreatitis severity and body composition characteristics (Table 2) Every oneLight Mildly intense P-value severity

TaxationIQR: 139.7–290.5; 144.9–283.6; 131.7–287.5; 172.1–333.5; Median: 217.3~221.2208.1~238.8~0.10a

A SAT median of 204.5, 213.5, 212.0, and 0.97a14.5-285.1 151.9-274.8 141.6-289.6 144.8-281.6

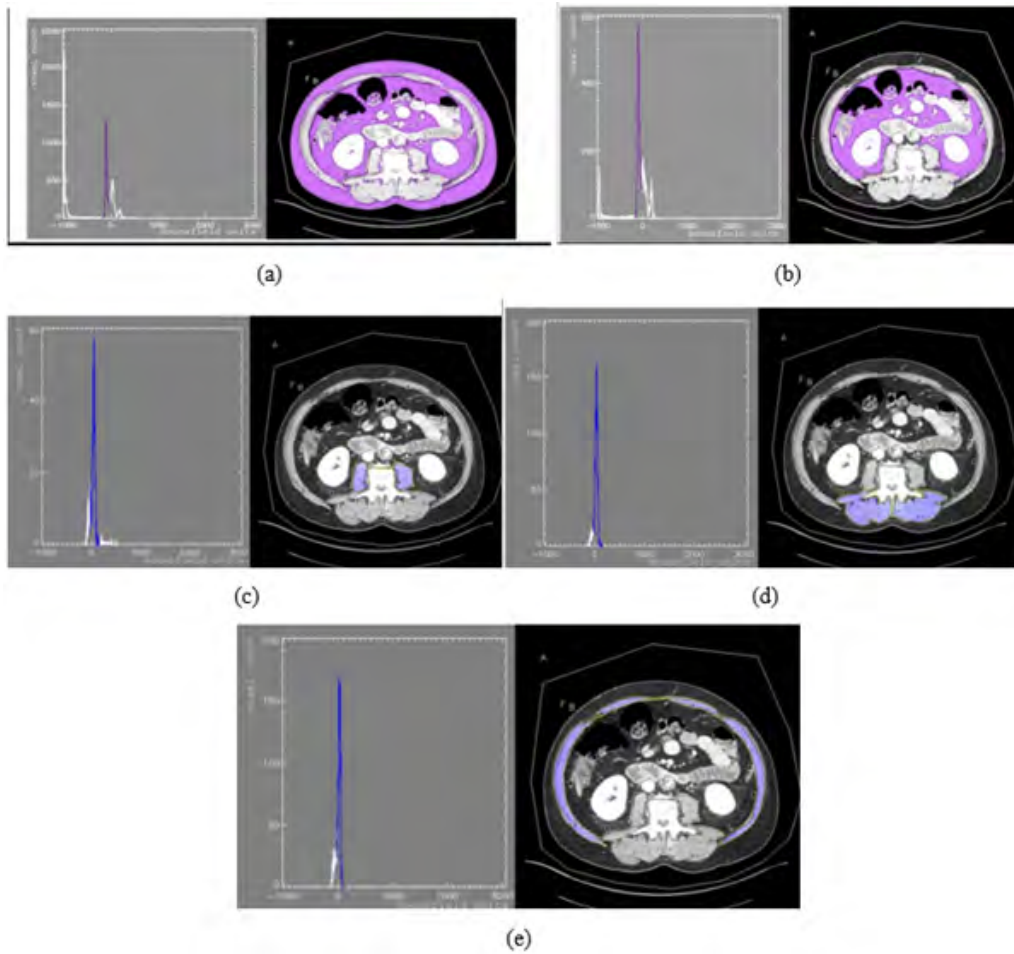
Median MM 129.5 126.6 130.0 135.6 136.9 0.037a 115.3–158.6 100.1–153.4 108.0–160.1 108.1–169.4 IQR

Median MMA 29.3 29.8 29.6 23.4 0.003aIQR~22.2–36.6 22.4–37.8 23.2–37.1 18.3–32.1

Median 1.64, 1.76, 1.57, 1.73, and 0.14a of VMRIQR<1.14–2.18 1.14–2.23 1.04–2.11 1.27:30–2.38

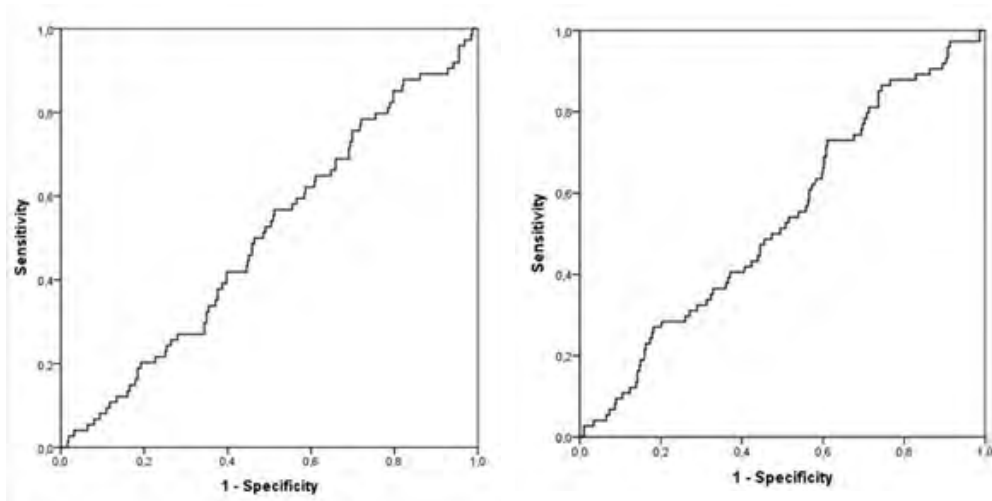
Note: MM = muscle mass; MMA = mean muscle attenuation; VMR = visceral muscle ratio; VAT, SAT, MM, and VMR in cm², MMA in HU. SAP is subcutaneous adipose tissue.

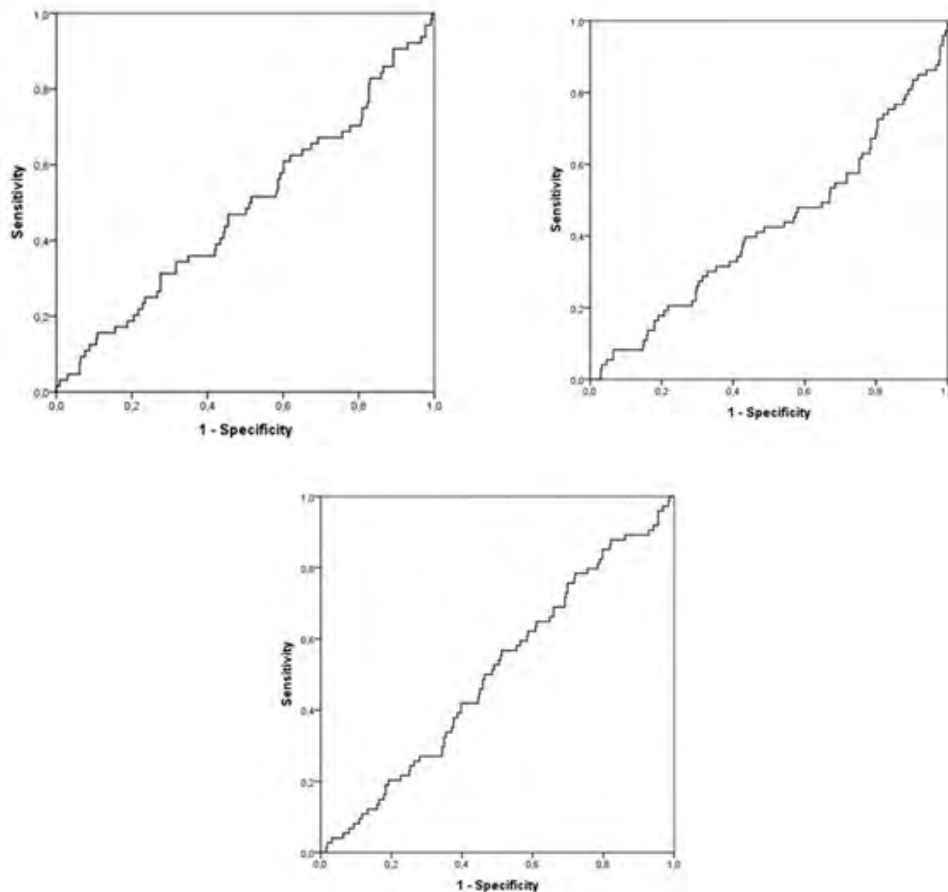
Kruskal–Wallis test.



Supplementary Figure 1: The DCMtool's analysis of the various body parts' regions of interest (ROI) Hounsfield units (HU), which range from -190 to -29 for fat and from -30 to +150 for muscles, were found in the voxels of the manually designated sections.

After that, the SAT, VAT, and MM were measured in centimeters squared using the area of the conforming voxels inside the CT slice. Each picture shows the ROI (CT slice on the right) as blue, and the other portion (left) shows a histogram of the voxel count for each HU, with blue indicating the range of interest. (A) shows the analyses of the visceral and subcutaneous fat, (B) just the visceral fat compartment, and (C–E) the studies of the muscle compartments. The average heart rate (HU) of the voxels tallied in each of the three muscle sections was used to calculate the mean muscle attenuation (MMA).





Supplementary Figure 2: ROC curves (receiver operating characteristic) and the corresponding areas Under the curves to forecast the severity of AP for muscle characteristics and body composition acquired from CT. The ROC curve for VAT is represented by (A), which has an area under the curve of 0.536; the corresponding values for (B), SAT, MM, MMA, and VMR are (0.484, 0.571, 0.424, and 0.503, respectively). Every region A p-value of < 0.05 was deemed statistically significant in all studies, yet the curves were tiny and no cut-off value for severe AP could be found. noteworthy

4.5. The Impact of AP on the Corporal Parameters

This has been the subject of further investigation over time. We compared the body parameter data for 41 successive CT scans (first vs. second CT). There were notable variations in the moderately severe group for MM (MMearly mean = 131.2 cm² vs. MMlate mean = 123.8 cm²; p-value 0.0003) and VAT (VATearly mean = 240.1 cm² vs. VATlate mean = 220.5 cm²; p-value 0.0083); refer to the Supplementary Residual Analysis Table 1.

Table 3 provides an overview of the findings from age- and sex-adjusted univariate and multivariate regression analyses. BMI, age, and sex were not associated with severe AP in either of the analyses. The lowest tertile (OR = 1) was used as a reference point for the ORs of VAT and SAT. Because we believed that lower VAT and SAT would be beneficial for the development of severe AP, this strategy was selected. A larger percentage of muscles is also anticipated to be advantageous for the duration of illness. Therefore, the reference tertile (OR = 1) was highest for the muscle metrics MM, MMA, and VMR. There was no significant correlation between

most indicators and severe illness. For patients in the highest tertile of VAT (OR 2.24, p = 0.042) and the lowest tertile of MMA (OR 3.446, p = 0.0004), univariate analysis revealed a significant association with severe AP. The link persisted for the MMA tertile in the multivariate analysis (OR 4.09, p = 0.003). Patients in the MM mid-tertile group had a significantly lower risk of developing severe AP (OR 0.496, p-value 0.038).

The univariate and multivariate regression analyses of risk factors for severe acute pancreatitis using univariate and multivariate regression are shown in Table 9.

Ninety-three patients were in the highest tertile in the interval between the two age groups.

Probability factors for severe AP by logistic regression analysis OR (95% CI) for univariate (p-values) All-comers a OR (95% CI) p-values

sex First female (ref) Men 1.126 (0.661–1.920) 6.662–2.17 (0.682–2.172) 0.505 Years of Age **** 73 Reference number one: 73 years Just 1.609 (0.916–2.824) [0.098<1.495 (0.815–2.745)]

BMI of 0.194 $\lt; 25.0$~1 (doc)1.86-34.9 0.998 (0.517-1.863) (0.512-0.981; 0.517-1.863) 0.954 $\lt; 18.5$-0.001 (0.000-0.002) It is 0.001$\lt; 0.001$ (0.000-0.002). 0.001 Int.TOP TERTIABLE In 2.242 (1.031-4.876), It is 0.042$\lt; 2.157$ (0.953-4.883). Half tertile, 0.065Amounts: 1.569 (0.761-3.234)~0.222~1.551 (0.750-3.207) 1 (ref) SAT 0.236 Lowest tertile Greater than 0.923 (0.420-2.027) 8841-2921 (0.395-2.148) 0.848 [Centile 0.994 (.508-1.946) in the middle 0.987-9.882 (0.499-1.932) Minimum tertile 1 (ref) = 0.958 MM Maximum tertile: 1 (ref) ^ Mid-tertile: 0.596 (0.324-1.094) [0.095$\lt; 0.496$] (0.256-0.961) * Lowest tertile: 0.038; range: 0.306-1.094 [0.194-1.126] = 0.199~0.432M MMA of 0.085 The mid-tertile, 1.536 (0.670-3.519), is the highest tertile, 1 (ref). 1.577-8.680 = 0.311~1.5770.286%(1.468-8.086) Lowest Tertile:3.4460.0004 4.085-1.609 (10.37) Highest tertile:1 (ref); mid-tertile:0.921 (0.494-1.714); 0.003 VMR0.594%~1.02 (0.538-1.954) Bottom

tertile:0.940 0.587 (0.268-1.287) 0.184 0.673 (0.300-1.508) 0.333 Note: = OR; = mean muscle attenuation; = visceral muscle ratio; MM = muscle mass; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue.

Age and sex adjustments were made to the findings of the multivariate analysis.

(Supplementary Table 1s: Multivariate analysis of corporal parameters by regression adjusted for etiology, age, sex, and CT time (days)

4.6. Curves of ROC

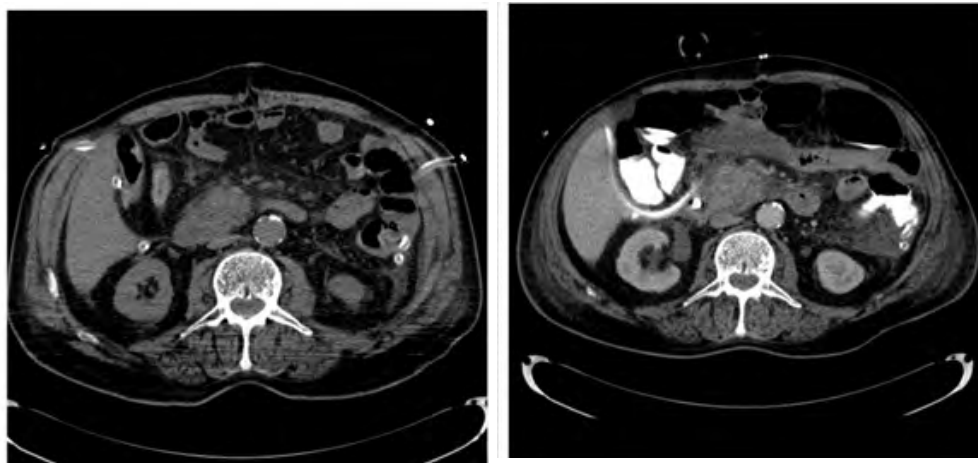
The predictive power of each parameter was analyzed using an ROC curve (Figure 3 Supplementary, A-E). A cutoff value for severe AP could not be determined, and the areas under the curves were small (range, 0.424-0.571).

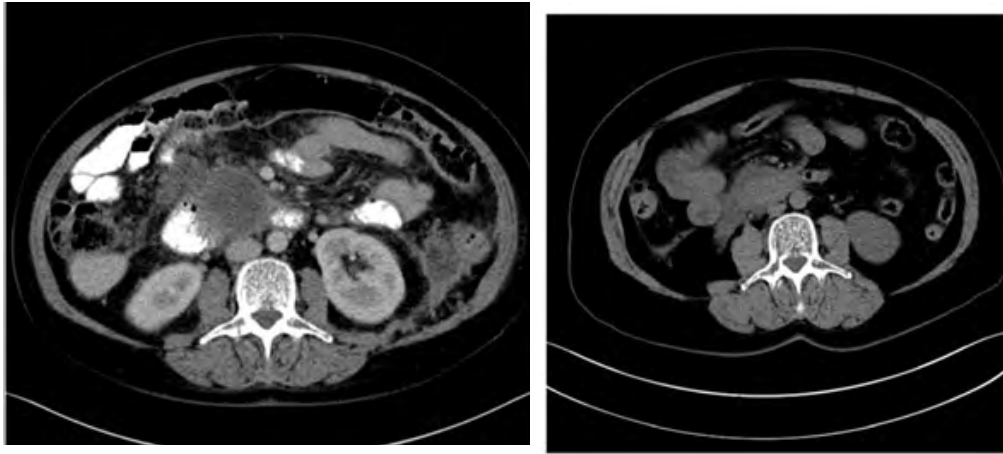
Supplementary Table 1: Multivariate analysis of corporal parameter by regression; adjusted for aetiology, age, gender, and CT time (days).

Severity	Cases No	Timing of Ctmedian (Range)	VAT cm2 mean (median)	SAT mean median	MM mean (median)	MMA Mean Median	VMR mean Median
Mild	7	2(1-3)	223.9 (219.8)	309.7 (318.6)	112.6 (113.0)	23.2 (22.2)	2.11 (1.83)
		12 (7-16)	231.6 (233.2)	288.0 (277.5)	103.9 (98.29)	24.9 (24.7)	2.33 (1.88)
Moderately Severe	23	3 (1-7)	240.1 (228.8)	240.8 (226.6)	131.2 (121.7)	27.6 (29.6)	1.83 (1.66)
		12 (8-16)	237.9 (235.0)	123.8 (121.2)	123.8 (121.2)	28.0 (28.2)	1.76 (1.66)
Severe	11	2 (1-6)	218.4 (184.8)	157.9 (142.8)	157.9 (142.8)	27.6 (25.6)	1.58 (1.36)
		13 (9-17)	215.9 (220.1)	152.1 (144.1)	152.1 (144.1)	22.8 (22.0)	1.52 (1.29)
Overall	41	2 (1-6)	246.6 (204.1)	135.2 (123.6)	135.2 (123.6)	26.9 (29.0)	1.81 (1.72)
		12 (7-17)	240.6 (235.0)	128.0 (121.2)	128.0 (121.2)	26.3 (26.8)	1.79 (1.66)

To determine whether measured body parameters are altered during the course of AP we compared consecutive CTs where available. Timing of the CTs represents the day after admission when the CT has been performed.

For the comparison the Wilcoxon T-test was applied. Overall VAT (p=0.0083) and MM (p=0.0003) was significantly altered by the disease course, whereas MMA did not differ (p=0.215). For VAT and MM a significant difference was observed for the moderately severe cases (p=0.0022; p=0.0288). SAT and VMR differed significantly in mild cases during the disease course (p=0.018 for SAT and VMR). * Significant differences (p<0.05).





Supplementary Figure 3: Two examples of sequential CT scans demonstrating the impact of the thoracic cavity's volume, bowel distension, and fluid collections all of which are the result of AP itself on the VAT measurement. One patient's initial CT scan is displayed in A and C, while the subsequent CT scans of the same patient are displayed in B and D. VAT in B 129.1 cm², D 166.8 cm², and A 190.3 cm² and C 129.8 cm².

5. Discussion

In contrast to the prognostic factor score and age, visceral obesity had no appreciable additional effect on mortality in 238 Egyptian SAP patients. There was also no appreciable difference in survival; however, visceral obesity may lengthen the ICU stay. Nine of the 11 studies demonstrated a strong correlation between VFA and extent according to a recent systematic literature review. However, only two out of 11 studies found an important link between VFA and mortality, and the remainder of the research either found no link or had insufficient data to conclude [8]. Therefore, it is not yet known whether VFA can affect mortality in individuals with AP; however, it does not appear to have a significant effect.

Numerous investigations have demonstrated that being overweight, BMI, and intra-abdominal fat on CT are independent risk factors for SAP severity, regional consequences, or death. Additionally, a few recommendations have demonstrated that being overweight is a significant prognostic factor [6, 7, 12-14] and that BMI > 30 kg/m² increases mortality and the likelihood of developing fatal illnesses [9, 16]. Very few individuals in Asian nations have BMI values that are too high; there did not appear to be a correlation between high BMI and SAP, and even taking BMI into account did not improve the ability to predict the severity of AP [17]. A national study conducted in Japan found that 25 of 825 individuals with AP had a BMI > 30 kg/m² (3%), and among these obese patients, only one fatality occurred [18]. Only 12 patients (5%) in the current study had a BMI > 30 kg/m², and they failed away. As the Egyptian Society for the Study of Obesity defines a BMI > 25 kg/m² as obese for the Egyptian population based on numerous epidemiological studies, we also compared 50 patients with a BMI > 25 kg/m² and 188 with a lower BMI, which suggested little variation in mortality (4.0 vs. 8.5%, P = 0.6825). Additionally, the BMI COV (20.5) demonstrated a poor predictive ability for SAP fatalities. We concluded that compared with the prognosis factor score, greater BMI had no significant effect on mortality risk

in Egyptian patients with SAP.

There is disagreement regarding the significance and effect of intra-corporal adipose tissue distribution on AP severity [3]. Significant discrepancies between our results and those from earlier studies on VAT and SAT were discovered. The median SAT was 209.2 cm², and the median VAT was 217.3 cm². The groups with mild (median levels 80–105 cm²) and moderately severe (median levels 89–150 cm²) AP also had lower levels of VAT and SAT, according to several studies that showed an association between adipose tissue characteristics and severe AP [10-12]. Although difficult to explain, this clear variation when compared to our data is intriguing. The cited studies examined two Asian and one American populations with varying etiologies, employed a range of software tools for their investigations, and did not adhere to the same AP classification. Our findings are in line with a recent publication from the Netherlands that examined the relationship between CT-assessed body composition and mortality in patients with necrotizing pancreatitis as well as an Australian study on patients in intensive care. These findings are consistent across Europe [16,17-32]. Given that our approach is comparable to that of the Dutch study and that there is a significant discrepancy in the median VAT and SAT levels between American and Asian studies, we hypothesized that the observed variations are due to regional or ethnic disparities. In terms of adipose tissue parameters, several studies [15,16,18] presented findings that are comparable to ours; however, the examined outcomes and setup of these publications vary, making it challenging to directly compare our findings with theirs.

We questioned whether AP itself would have an impact on VAT assessment since we saw an association between severity and VAT in univariate analysis (OR 2.24; 95% CI 1.03–4.88; p-value 0.04) but not in multivariate analysis (p = 0.07). The findings for the moderately severe group showed a substantial difference in the VAT and MM, as shown in Supplementary Table 1 and Supplementary

Figure 3. Given the comparable tendency in the SAT group, the gradual decline in VAT over time in both the moderately severe and severe groups may reflect a general disease-induced reduction in adipose tissue. When the first and second CT scans were directly compared, it became clear that factors such as intestinal distension, thoracic cavity volume, and fluid collection caused by AP affected the assessment of VAT. Additionally, MM showed a general drop, which may have been caused by hospitalized and unwell patients who were in a state of general edema and reduced muscle mass. We were able to partially recreate the link between MM depletion observed in a previous study from the Netherlands during the course of the disease in our subsequent CTs. Nevertheless, prospective trials with predetermined protocols are required, because it is challenging to draw generalizations regarding such cases based on retrospective data.

A VFA exceeding 100 cm² was detected in 76 (57%) patients in the current investigation, which was not greater than the 62% of 5347 asymptomatic Egyptian individuals with a mean age of 54 years [21]. Patients with VFAs less than 100 cm² and obese patients experienced similar death rates (8.8% vs. 5.9%, $P = 0.7307$). There was no difference when using multivariate analysis and the log-rank test, even though a higher VFA value of 167 cm² was used to predict mortality, which revealed a substantially higher OR of 4.38 ($P = 0.0406$) for predicting mortality in the univariate logistic analysis. However, when a greater VFA was employed as the COV, a greater VFA exhibited a marginally significant mortality risk compared to a lower VFA (19.1% vs. 5.1%, $P = 0.0505$), which may be related to the small patient population.

The single-hospital retrospective design of this study has some limitations. Initially, few patients with SAP were included in the multivariate analysis or ROC curve, which may have hampered the ability of the study to generalize its findings and lead to overfitting. The VFA evaluated by CT in the present investigation had no discernible extra effect on death, whereas SAP participants with a VFA of 167 cm² or more died at an increased rate compared to SAP individuals with a VFA of less than 167 cm². Further SAP cases should be analyzed to reveal the effect of VFA on mortality. As a result, even though VFA may be underestimated, it does not appear to have a greater effect than prognostic factor score or age. Furthermore, because Japanese guidelines are regularly used in medical practice, we assessed the practice of AP using the Japanese seriousness standards for AP rather than the Acute Physiology and Chronic Health Evaluation II (APACHE II) score [22] or the modified Marshall scoring system, which is supported by the revised Atlanta classification [23]. The prognostic factor score has the same potential to predict mortality as the APACHE II score, according to a new multicenter validating research involving 1159 Japanese AP patients.[24] The results of this validation investigation also revealed that the updated Atlanta classification's diagnostic factor score had an AUC of 0.83 (95% CI, 0.81-0.86) to forecast

seriousness, a value that was considerably greater than APACHE II's AUC of 0.80 (95% CI, 0.77-0.82) ($P = 0.001$). Consequently, we believe that it is possible to assess the role of visceral fat in predicting death in Egyptian AP patients using the prognostic factor scores of the Japanese recommendations. Third, we only used the cord-level VFA data; we did not use the overall volume or L3 level VFA data. Owing to its complicated nature, applying the complete volume is impractical, and because the umbilical level is the same as the waist circumference, it delivers the highest ratio of fat mass to overall body size.[25] The COV of VFA at the L3 level, which was 158 cm², was also assessed and its AUC was comparable to that at the umbilical level (AUC:0.669) (Table 3). Therefore, umbilical fat was measured.

There has not been much research on how muscle parameter levels affect the AP. According to our data, instances of severe AP (136.9 cm²) had a higher MM than cases of moderately severe (130 cm²) or mild (126.6 cm²) AP (p -value = 0.037). These figures indicate that low MM is typically associated with worse disease outcomes. One theory is that patients who were critically ill, even at an early stage of the disease, had greater fluid sequestration, and that edema affected the measurement as a result, producing erroneously high numbers. Similarly, lower MM levels were surprisingly linked to less severe AP in the multivariate analysis; however, this was only significant for the mid-tertile ($p = 0.038$). The p -value for the lowest tertile of MM was 0.085, indicating that fewer patients in this cohort generated Type 2 errors. As we believed that the highest tertile of MM would yield a more favorable outcome, we used it as a reference. Even after adjusting for age and sex, our results showed the reverse, which led us to believe that edema may once again be a contributing factor. We have not been able to find any alternative hypothesis to compare this with nor can this theory be verified in this investigation. Only individuals with severe AP were examined in Brewster et al.'s work, and their level of MM was 167.8 (144.2–203.7) cm², which is consistent with our data [17]. Although the Australian study subjects were younger, no information was provided on the exact day the CT scan was performed throughout the patient's illness.

Regarding patients examined for muscle mass index effect on the severity of acute pancreatitis, the study showed a substantial correlation between severity and a low level of MMA, which indicates myosteatosis (aOR 4.1, 95% CI 1.6–10.4, p -value 0.003). Furthermore, the disease course had no effect on measurements (Supplementary Table 1; $p = 0.215$). Furthermore, a study by van Grinsven et al., which examined the relationship between MMA and mortality in patients with AP, provided some support for this observation. The researchers found a significant association in the univariate analysis (OR 2.37, 95% CI 1.43–3.92, p -value 0.001) but not in the multivariate analysis (OR 1.13, 95% CI 0.62–2.08, p -value 0.69). Intramuscular fat content, as opposed to intermuscular fat content, defines the MMA. Previous research has linked low muscle

attenuation to a worse prognosis as well as an increased chance of complications and morbidity in cancer patients. Although the makeup of intramuscularly deposited fat is unknown, it has been suggested that fat infiltration causes decreased muscle attenuation. Myosteatosis has been linked to various clinical illnesses; however, the underlying mechanisms are not well understood, making it difficult to understand how it affects the severity of AP.

Our study has several limitations. The study's retrospective design suggested that despite the fact that all centers employed identical standard protocols for their pancreatic research, there was no precise predetermined study protocol for CECTs. Furthermore, individuals with a moderate disease course who did not undergo CECT were not enrolled, which may have introduced selection bias. Only patients who underwent CECT were included in the study. Unless another diagnosis needs to be ruled out, patients with mild illnesses do not require CECT according to AP guidelines [27]. As a result, compared to studies where all AP patients were included consecutively, the group of patients with mild AP is probably smaller in radiology research, and the number of patients with moderately severe illnesses is higher [28]. We also note that there is still much to learn about how oedema affected our findings and every other study on body parameters in AP. However, we assembled a sizable and accurately characterized AP cohort from locations across Europe, providing our study with a strong research approach. Furthermore, there is a lower chance of disproportion in terms of baseline patient features and cultural variance in the pan-European multicenter scenario.

Ultimately, our data do not support previous research, suggesting that specific visceral fat parameters or obesity are generally linked to the severity of AP. According to previous research, people with low MMA are associated with more severe disease, but those with low MM have better outcomes. Further research is necessary to fully understand the mechanisms underlying these results. Since many of our findings and those from other studies come from looking back, it seems to be the best way to solve problems that have not yet been solved would be to perform a similar but prospective study with patients from different parts of the world. Finally, we believe that one of the most important prerequisites for a thorough understanding of the pathophysiological mechanisms of severe AP is the awareness of the role and impact of adipose tissue within the abdominal cavity. For the Egyptian community with SAP, we observed that abdominal fat being overweight, which was assessed by VFA at the umbilical level, had no substantial supplementary effect on predicted death compared to the forecasting predictor value and ageing.

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